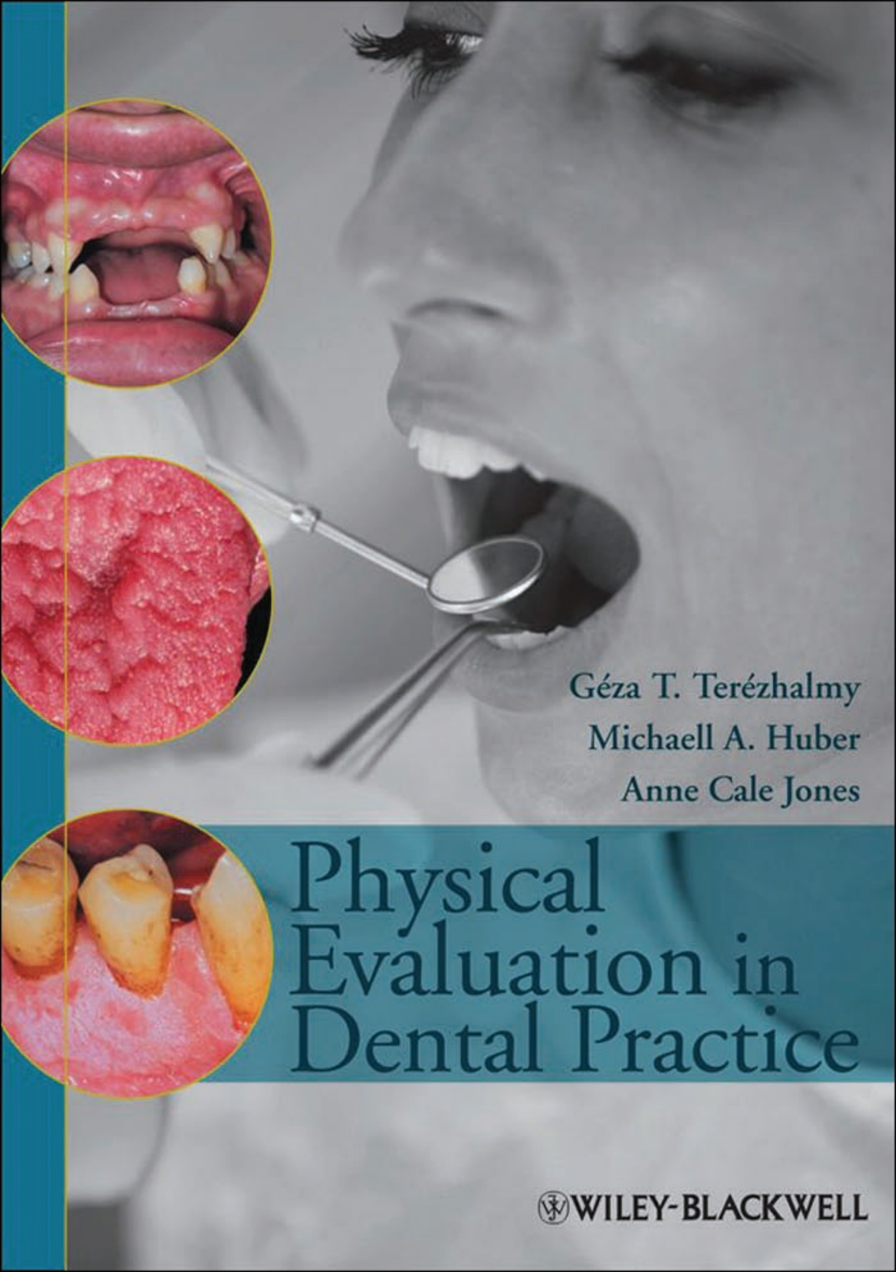

Géza T. Terézhalmy
Michaell A. Huber
Anne Cale Jones
Physical Evaluation in Dental Practice
WILEY-BLACKWELL

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with contributions by Vidya Sankar and Marcel E. Noujeim



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Géza T. Terézhalmy is Professor and Dean Emeritus at the School of Dental Medicine, Case Western Reserve University, in Cleveland, Ohio. **Michaell A. Huber** is Associate Professor and Head of the Division of Oral Medicine in the Department of Dental Diagnostic Science at the University of Texas Health Science Center at San Antonio Dental School. **Anne Cale Jones** is Professor in the Department of Pathology at the University of Texas Health Science Center at San Antonio Dental School.

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Preface

> Learn to see, learn to hear, learn to feel, learn to smell, and know that by practice alone can you become an expert.
>
> Sir William Osler

Diagnosis is the bridge between the study of disease and the treatment of illness. Making a distinction between disease and illness appears redundant because the words frequently are used interchangeably. However, diseases of the oral cavity and related structures may have profound physical and emotional effects on a patient, and a holistic approach to patient care makes this distinction significant. In oral pathology one studies disease; in clinical dentistry one treats illness. For example, necrotizing ulcerative gingivitis may be defined with special emphasis on the microbiological aspects of the disease, or one may speak of an inflammatory reaction featuring "punched-out" erosions of the interdental papillae. However, necrotizing ulcerative gingivitis is more complex. It is the totality of symptoms (subjective feelings) and signs (objective findings) that together characterize a single patient's reaction—not merely a tissue response—to infection by spirochetes. While disease is an abstraction, illness is a process.

Similarly, clinicians must recognize that systemic disease may affect the oral health of patients and to treat dental disease as an entity in itself is to practice a rigid pseudoscience that is more comforting to the clinician than to the patient. The diagnosis and treatment of advanced carious lesions afford little support to the patient if one overlooks obvious physical findings suggesting that the extensive restorative needs were precipitated by qualitative and quantitative changes in the flow of saliva secondary to an undiagnosed or uncontrolled systemic problem, or anticholinergic pharmacotherapy. The clinician with a balanced view of dentistry will recognize that caries is only a sign of disease and preventive and therapeutic strategies will have to be based on many patient-specific factors.

It is axiomatic that while dentists are the recognized experts on oral health, they must also learn of systemic diseases. Such an obligation is tempered only by the extent to which systemic diseases relate to the dental profession's anatomic field of responsibility, the extent to which illnesses require modification of dental therapy or alter prognoses, and the extent to which the presence of certain conditions (infectious diseases) may

affect caregivers. Consequently, clinicians should not treat oral diseases as isolated entities. They should recall that physical signs and symptoms are produced by physical causes. Since physical problems are the determinants of physical signs and symptoms, these signs and symptoms must be recognized before the physical problems can be diagnosed and treated.

It is through the clinical process that clinical judgment is applied and, with experience, matures. Clinical judgment does not come early or easily to most clinicians. It is forged from long hours of clinical experience and a life-long commitment to the disciplined study of diseases and illnesses. Clinicians should study books to understand disease, study patients to learn of human nature and illness, and model mentors to develop clinical judgment. Ultimately, the experienced clinician will merge the science of understanding disease and the art of managing illness. These activities should be fostered by the clinician's sincere desire to minimize patient discomfort, both physical and emotional, and to maximize the opportunities to provide optimal care.

Contributor List

Géza T. Terézhalmy
Professor and Dean Emeritus
School of Dental Medicine
Case Western Reserve University
Cleveland, Ohio

Michaell A. Huber
Associate Professor
Head, Division of Oral Medicine
Department of Dental Diagnostic Science
The University of Texas Health Science Center at San Antonio Dental School
San Antonio, Texas

Anne Cale Jones
Professor
Department of Pathology
University of Texas Health Science Center at San Antonio Dental School
San Antonio, Texas

Vidya Sankar
Assistant Professor
Division of Oral Medicine
Department of Dental Diagnostic Science
University of Texas Health Science Center at San Antonio Dental School
San Antonio, Texas

Marcel E. Noujeim
Assistant Professor
Director, Graduate Program
Division of Oral and Maxillofacial Radiology
Department of Dental Diagnostic Science
University of Texas Health Science Center at San Antonio Dental School
San Antonio, Texas

Physical Evaluation in Dental Practice

1 Introduction to the Clinical Process

Patients consult clinicians to obtain relief from symptoms and to return to full health. When cure is not possible, intervention to improve the quality of life is warranted. Consequently, oral healthcare providers' primary obligation is the timely delivery of quality care within the bounds of the clinical circumstances presented by patients. The provision of quality care will depend on timely execution of the clinical process.

Essential Elements of the Clinical Process

The clinical process represents a continuous interplay between science and art and may be conveniently divided into three phases.

Phase I

Phase I of the clinical process is physical evaluation and consists of eliciting a historical profile, performing an examination, obtaining appropriate radiographs, ordering laboratory tests, and, when indicated, initiating consultations with or referrals to other healthcare providers. The information obtained is systematically recorded. In order to optimize the yield, clinicians need to possess an inquiring mind, discipline, sensitivity, perseverance, and patience.

Phase II

Phase II of the clinical process involves an analysis of all data obtained during Phase I. Interpretation and correlation of these data, in the light of principles gained from the basic biomedical and clinical sciences, will create the diagnostic fabric that will lead to a coherent, defendable, relevant, and timely diagnosis. This is an intellectual and, at times, intuitive activity. In making diagnoses, clinicians must recall their knowledge of disease.

Phase III

Phase III of the clinical process is centered around the timely development and implementation of necessary preventive and therapeutic strategies and communicating these strategies to the patient or guardian in order to obtain consent and to encourage compliance with and participation in the execution of the plan. In deciding on management strategies, clinicians must think in terms of illness and the total impact of a disease on a given patient and his or her immediate family.

Quality Management in the Clinical Process

A four-part control cycle (plan-do-check-act) introduced to industry in the 1930s is applicable to total quality management (TQM) in the clinical process and is reflected in the acronym CEAR (pronounced CARE): criteria-execution-assessment-response. Criteria are intended to maintain established standards. Ideally, standards should be based on knowledge derived from well-conducted trials or extensive, controlled observations. In the absence of such data, they should reflect the best-informed, most authoritative opinion available. Execution is the implementation of activities intended to meet stated standards. Assessment is comparing the impact of execution (outcome) against the stated standards. Response refers to the activities intended to reconcile differences between stated standards and observed outcome (Table 1.1).

TQM provides the fabric for a disciplined approach to work design, work practices, and constant reassessment of the clinical process. In TQM there is no minimum standard of "good enough"; there is only "better and better." Defects are signals that point to parts of a process that must be improved so that quality is the result.

Table 1.1. Activities intended to correct a problem identified by the control cycle.

Reconsider the criteria (standard).
Redesign the activities intended to achieve the criteria
Review the assessment process.
Remediate without changing the criteria or the activities intended to achieve the criteria.
Reject the samples that do not meet the criteria.
Apply residual learning to the next control cycle.

Factors Affecting Quality

Amenities of Care

The amenities of care represent the desirable attributes of the setting within which the clinical process is implemented. They include convenience (access, availability of service), comfort, safety, and privacy. In private practice these are the responsibilities of the clinician. In institutional settings, the responsibility lies with the administrators of the institution.

Performance of the Clinician

The clinical process is a combination of intellectual and manipulative activities by which disease is identified and illness is treated. As we seek to define its quality, we must consider the performance of clinicians. There are two elements in the performance of clinicians that affect quality, one technical and the other interpersonal.

Technical performance depends on the knowledge and judgment used in arriving at appropriate diagnostic, therapeutic, and preventive strategies and on the skillful execution of those strategies. The quality of technical performance is judged in comparison with the best in practice. The best in practice, in turn, has earned that distinction because it is known or is believed to lead to the best outcome. The second element in the performance of the clinician that affects quality is interpersonal skills (see "Patient-Doctor Communication in the Clinical Process").

Performance of the Patient

In considering variables that affect the quality of the clinical process, contributions made by the patient, as well as by family members, must also be factored into the equation. In those situations in which the outcome of the clinical process is found to be inferior because of lack of optimal participation by the patient, the practitioner must be judged blameless.

Assessing Quality

Effective control over quality can best be achieved by designing and executing a clinical process that meets professional standards and also acknowledges patients' expectations. The information from which inferences can be drawn about quality may be classified under three headings: structure, process, and outcome.

Structure

In addition to the amenities of care discussed earlier, structure also denotes the attributes of material resources (e.g., facilities and equipment), human resources (e.g., the number and qualification of personnel), and organizational resources (e.g., convenience [access, availability of service], comfort, safety, privacy, methods of payment). Since structure affects the amenities of the oral healthcare setting, it can be inferred that good structure increases the likelihood of a good process.

Process

Process denotes what is actually done in the clinical process. It includes the clinician's activities in developing and recommending diagnostic, therapeutic, and preventive strategies; and the execution of those strategies, both by the clinician and the patient. Process also includes the values and virtues that the interpersonal patient-doctor relationship is expected to have (i.e., confidentiality, informed consent, empathy, congruence, honesty, tact, and sensitivity). In general, it can be assumed that a good process increases the likelihood of good outcome.

Outcome

Outcome denotes the effects of the clinical process on the identification and treatment of consequential problems, improvement in health, and changes in behavior. Because many factors influence outcome, it is not

possible to determine the extent to which an observed outcome is attributable to an antecedent structure or process. However, outcome assessment does provide a mechanism to monitor performance to determine whether it continues to remain within acceptable bounds.

Patient-Doctor Communication in the Clinical Process

Poor skills in communicating with patients are associated with lower levels of patient satisfaction, higher rates of complaints, an increased risk of malpractice claims, and poorer health outcomes. Clearly, in the clinical process, the performance of clinicians as it relates to interpersonal skills is the very source of their vulnerability. The process of establishing a patient-doctor relationship, however, is not easy. To illustrate this point, let us consider the clinical process in dealing with a patient in pain, the most common complaint causing a person to seek the services of an oral healthcare provider.

Ideally, the clinician should initiate the clinical process in a quiet, comfortable, private setting and foster a warm, friendly, concerned, and supportive approach with the patient. However, this may be a challenging task since it is well established that many patients experience anticipatory stress in the oral healthcare setting. Such stress may provoke patients to experience a state of disequilibrium or crisis characterized by anxiety, that is, an intense unpleasant subjective feeling and an inability to function normally. The sequence of events, which leads from equilibrium to a crisis situation (disequilibrium) and back to equilibrium, includes a hazardous event, a vulnerable state, a precipitating factor, an active crisis state, and a reintegration state.

Hazardous Event

A hazardous event is any stressful life event that taxes the patient's ability to cope. The experience can be either internal (the psychological stress of dental phobia) or external (such as a natural disaster, the death of a loved one, or the loss of employment). Clinicians may be unaware of such hazardous events and patients may not readily volunteer such information.

Vulnerable State

Depending on subjective interpretation, one person may see the hazardous event as a challenge, while another may see the same event as a threat. If one views the event as a threat, the increased physical and emotional tension may manifest itself as perceptions of helplessness, anxiety, anger, and depression.

Precipitating Factor

The precipitating factor (in our example, pain) is the actual event that moves the patient from the vulnerable state to the active crisis state. This event, especially when added onto other stressful life events (hazardous events), can cause a person to suffer a crisis. In susceptible patients, not only pain but even minor dental problems requiring a visit to the dentist can precipitate an active crisis state.

Active Crisis State

During the active crisis state, the patient is emotionally and psychologically aroused because of pain, negative self-critical thoughts about what brought him or her into the clinician's domain, unfamiliarity with the environment, and fear that the clinician will be judgmental or punitive. The model for crisis

intervention has six characteristic phases and follows the acronym CRISIS: calm confidence, responsiveness, involvement, supportiveness, “I can” statements, and situation.

Calm Confidence

People who are in a crisis situation generally are not attuned to the words being spoken to them, but they are responsive to nonverbal communication. Behaviorally, calm confidence is displayed by establishing eye contact with the patient, by guiding the patient into the chair, or by touching the patient’s shoulders. All of these measures reflect inner self-confidence and control over the situation. If the clinician is perceived as being calm and confident, the patient is more likely to calm down and give trust and control to the clinician.

Responsiveness

Responsiveness is conveyed through verbal communication. It requires a willingness to be directive and to give firm guidance while responding to both the emotional and oral healthcare needs of the patient. The clinician with empathy for the patient does not convey a negative value judgment and, therefore, builds rapport with the patient.

Involvement

A patient in crisis will exhibit behaviors suggesting helplessness or dependency, which might make the clinician feel all the more responsible. Clinicians must relinquish this sense of total responsibility and assist the patient to assume responsibility for his or her own health. The clinician can redirect responsibility by telling patients that their active involvement is needed for a successful long-term outcome. Positive encouragement increases the likelihood that patients will adopt the behaviors necessary to maintain their oral health.

Supportiveness

Listening to the patient relating his or her feelings, concerns, and experiences is a large part of being supportive. Expressing acceptance in a nonjudgmental style, such as sitting near the patient at eye level and nodding in an understanding manner, further conveys support. This does not imply that the clinician must agree with the ideas of the patient, but it does reflect a sense of support and concern for the patient.

″I Can″ Statements

Individuals often aggravate a crisis situation by expressing negative thoughts such as “I can’t handle this,” “This is too much for me,” or “I know this is going to be terrible.” Here, the clinician’s response may go a long way in determining a patient’s success in developing coping skills. By saying nothing, the clinician tacitly agrees with and reinforces an unhealthy line of thinking. On the other hand, by teaching the patient to use positive self-statements, the clinician helps foster healthy coping skills. Examples of positive coping thoughts include “One step at a time,” “I can handle this situation,” or “I can handle this challenge.” By positively confronting a crisis situation, the patient experiences less distress and is more responsive to intervention.

Situation

The situation is the crisis of the moment, and it reflects the physical and emotional state of the patient at that moment in time. It must be kept in mind that patients do not consult clinicians to obtain diagnoses, but to obtain relief from symptoms and to return to full health. When a cure is not possible, intervention to improve the quality of life is warranted. Successful resolution of the problem is often directly dependent on timely intervention. The situational component of the crisis mandates that the intervention produce both short-term and long-term results (Table 1.2).

Table 1.2. Primary goals of crisis intervention in the oral healthcare setting.

Identify the problem.
Establish a working diagnosis.
Restore function (at least temporarily).
Develop a plan for definitive treatment.
Help the patient to connect the current crisis with past ineffective behaviors.
Teach the patient new preventive healthcare skills.

Reintegration State

Reintegration refers to the transition back to equilibrium. Ideally, the patient feels that the clinician was responsive. The problem has been resolved in a timely fashion, function has been restored (at least temporarily), a plan for definitive treatment has been agreed upon, the current crisis has been successfully connected with past ineffective behaviors, and new preventive healthcare skills have been instituted.

Characteristics of the Patient-Doctor Relationship

Reflecting on the case of the patient in pain discussed above, it becomes clear that the characteristics that distinguish, promote, and maintain a healthy patient-doctor relationship are empathy, congruence, positive regard, and, as we shall see later, "due process."

Empathy

Empathy refers to the clinician's perception and awareness of the patient's feelings without participating in them. When the patient is sad, the clinician senses and acknowledges the sadness, but does not become sad. In contra-distinction, sympathy implies assumption of, or participation in, another person's feelings.

Congruence

Congruence relates to the matter of words and deeds conveying the same message.

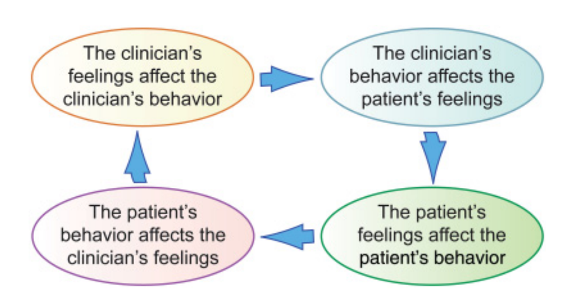


Figure 1.1. Clinician-patient interaction.

Patients will sense whether the clinician's words and deeds are congruent or convey divergent meanings. Similarly, if the patient says, "I am happy," but appears sad and dejected, the clinician should be alert to the discordant messages conveyed by what is heard and what is observed.

Positive Regard

Positive regard is the act of recognition and active demonstration to the patient that the clinician recognizes the patient as a worthy person. This means that the clinician makes a concentrated effort to get to know what the patient cares about; what makes the patient happy, sad, or angry; what makes the patient likable or unlikable; and identifies qualities that make the patient unique. In this process, the clinician transmits attitudes to the patient by the same unconscious word inflections, tones of voice, and body language by which the patient conveys underlying feelings to the clinician. The human qualities that the clinician and patient bring to the process of the patient-doctor interaction are crucial in either opening or closing the lines of communication (Figure 1.1).

Documentation of the Clinical Process

Attorneys, courts, and juries operate by the dictum "if it isn't written down, it didn't

Table 1.3. Essential elements of a progress note.

Database	Subjective data	The reason for the visit, a statement of the problem (chief complaint), and a qualitative and quantitative description of the symptoms as described by the patient.
	Objective data	"Measurements" (a record of actual clinical, radiographic, and laboratory findings) taken by the clinician undistorted by bias.
Problem list	Assessment	Derived from the database, which leads to a provisional or definitive diagnosis, i.e., "needs" (existing conditions or pathoses).
Disposition	Plan	Proposed treatment plan and actual services (preventive, therapeutic) rendered to alleviate or resolve problems: include plans for consultation or referral to other healthcare providers, prescriptions written, and pre- and postoperative instructions.

happen." Documentation of the clinical process should conform to state laws governing the practice of dentistry and the standards of care established by the American Dental Association and other relevant professional organizations.

Problem-Oriented Dental Record

Problem-oriented record keeping enjoys a significant degree of universality in both medical and dental settings. While there are many acceptable alternatives, the problem-oriented dental record facilitates the standardized sequencing of activities associated with the elicitation and documentation of demographic, diagnostic, preventive and treatment planning, and treatment-related information.

Progress Notes

Logically structured progress notes provide the fabric to effectively document and promote continuing problem-oriented patient care. They facilitate the chronological recording of all patient encounters and are divided into three main components: the database (subjective and objective data), the problem list, and the disposition of the problem (Table 1.3).

Table 1.4. The database.

- Patient identification
- Demographic data
- A statement of the problem
  - Chief complaint
    - Qualitative and quantitative description of the symptoms provided by the patient
  - Other reasons for the visit
    - New patient
    - Established patient
      - Recall
      - Emergency
      - Follow-up
- Historical profile
  - Dental history
  - Medical history
    - Family history
    - Social history
  - Review of organ systems
- Physical examination
  - Vital signs, height, and weight
  - Head and neck examination
  - Examination of the oral cavity
  - Radiographic studies
  - Laboratory studies
- Consultations
  - Dental
  - Medical
- Risk stratification

Database

The database is the product of those activities that are performed during Phase I of the clinical process (Table 1.4). These activities are

effective to screen for significant disease, and the results are likely to be good reference points in the evaluation of future problems. Consequently, screening measures should be validated and focused on identifying those problems that one cannot afford to miss.

An initial database is to be recorded on all new patients (Tables 1.5 and 1.6). The

Table 1.5. Documentation of initial historical profile.

NAME ______________________ ID NUMBER ______________________

Date of birth ______________________ Sex ______________________

Ethnic origin ______________________ Occupation ______________________

Address ______________________ City ______________________

State/Zip ______________________ Phone ______________________

Emergency contact Name ____________ Phone ______________________

Name ____________ Phone ______________________

Insurance information ____

CHIEF COMPLAINT

____

____

DENTAL HISTORY

Frequency of visits to dentist?

____

Date of most recent radiographic examination?

____

Types of care received?

____

History of oro-facial injury (date, cause, type of injury)?

____

Difficulties with past treatment?

____

Adverse reactions (local anesthetics, latex products, and dental materials)?

____

MEDICAL HISTORY

Drug allergies or other adverse drug effects?

____

____

____

Medications (prescribed, OTC, vitamins, dietary supplements, special diets)?

____

____

____

Past and present illnesses?

____

____

____

____

Last time examined by a physician (why)?

____

____

____

____

Females only (contraceptives, pregnancy, changes in menstrual pattern)?

____

____

____

Table 1.5. *Continued*

Family history (DM, HTN, heart disease, seizures, cancer, bleeding problems, other)?
____
____
____

Social history (type, amount, frequency of tobacco, alcohol, and recreational drug use)?
____
____
____

REVIEW OF ORGAN SYSTEMS

Skin
- Itching ________
- Rash ________
- Ulcers ________
- Pigmentation ________
- Lack/loss of body hair ________

Extremities
- Varicose veins ________
- Swollen, painful joints ________
- Muscle weakness, pain ________
- Bone deformity, fractures ________
- Prosthetic joint ________

Eyes
- Conjunctivitis ________
- Blurred vision ________
- Double vision ________
- Drooping eyelids ________
- Glaucoma ________

Ear, nose, throat
- Earache ________
- Hearing loss ________
- Nosebleeds ________
- Sinusitis ________
- Sore throat ________
- Hoarseness ________

Respiratory
- Shortness of breath ________
- Coughing, blood in sputum ________
- Bronchitis, emphysema ________
- Wheezing, asthma ________
- TB, or exposure to ________

Cardiovascular
- Hypertension ________
- Pain in chest, MI ________
- Congenital heart disease ________
- Prosthetic valve/pacemaker ________

Gastrointestinal
- Eating disturbance ________
- GERD, abdominal pain, PUD ________
- Liver disease ________
- Jaundice, hepatitis ________

Genitourinary
- Difficulty urinating ________
- Excessive urination ________
- Blood in urine ________
- Kidney problem ________
- STDs ________

Endocrine
- Thyroid problem ________
- Weight change ________
- DM ________
- Excessive thirst ________

Hematopoietic
- Bruising/bleeding ________
- Anemia ________
- White blood cell problems ________
- HIV infection ________
- Spleen problem ________

Neurological
- Headaches ________
- Dizziness, fainting ________
- Seizures ________
- Paresthesia/neuralgia ________
- Paralysis ________

Psychiatric
- Anxiety, phobia ________
- Depression ________
- Other ________

Growth or tumor
- Surgery ________
- Radiotherapy ________
- Chemotherapy ________

Table 1.6. Documentation of initial physical examination.

NAME ______________________ ID NUMBER ______________________

VITAL SIGNS, HEIGHT, AND WEIGHT

Blood pressure ______________ Pulse ______________

Respiration ______________ Temperature ______________

Weight ______________ Height ______________

HEAD AND NECK EXAMINATION

Head ______________

Face ______________

Facial bones ______________

Ears ______________

Nose ______________

Eyes ______________

Hair ______________

Neck ______________

Lymph nodes ______________

TMJ ______________

Salivary glands ______________

Neurological findings ______________

INTRAORAL EXAMINATION

Lips/commissures ______________

Mucosa ______________

Hard palate ______________

Soft palate/tonsillar area ______________

Tongue ______________

Floor of the mouth ______________

Gingivae ______________

Breath ______________

Teeth/occlusion/periodontal status (PSR)

PSR | | |

A B C D E F G H I J

Right 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 Left

32 31 30 29 28 27 26 25 24 23 22 21 20 19 18 17

T S R Q P O N M L K

PSR | | |

Remarks

____

____

____

documentation is to be made legibly and in ink. The use of symbols such as check marks and underlined or circled answers are best avoided. Responses to queries are to be recorded as "positive" (with appropriate elaboration), "negative," or "not applicable." The database is to be reviewed at all subsequent appointments and changes recorded in the progress notes of that day (Table 1.7).

Problem List

A problem is anything that requires diagnosis or treatment or that interferes with the quality of life as perceived by the patient. It may be a firm diagnosis, a physical sign or symptom, or a psychological concern. Problems by their nature may fall into one of several categories (Table 1.8).

A complete database is so essential to the success of the clinical process that clinicians must consider an "incomplete database" as the number one problem until all required data have been obtained. An incomplete database may provide the basis for initial consultation with, and referral to, dental and medical specialists. Subsequently, the resolution of diagnostic problems may lead to further consultations with, or referrals to, colleagues, other healthcare professionals, and allied healthcare workers (see chapter 8).

Disposition of the Problem

The clinical process culminates in the development of timely preventive and therapeutic strategies, along with the explanation of these strategies to the patient or guardian, in order to obtain consent and to encourage compliance with, and participation in implementing the treatment plan (see chapter 8).

Table 1.7. Progress notes.

NAME	ID NUMBER	
Date	PROGRESS NOTES	Signature
00/00/00		
S	Subjective data: reason for the visit; changes to the medical history	
O	Objective data: "measurements" taken by the clinician (clinical, radiographic, and laboratory data; vital signs)	
A	Assessment: diagnosis derived from subjective and objective data (reason for therapeutic intervention)	
P	Plan: treatment plan or actual treatment provided; prescriptions written; postoperative instructions; disposition	
		Signature

Table 1.8. Problem categories with examples.

Anatomic (developmental, acquired)	Psychiatric (anxiety, depression)
Physiological (pallor, jaundice)	Abnormal diagnostic tests
Symptomatic (pain, dyspnea)	Risk factors (heart disease)
Physical (paralysis)	Socio-economic (uninsured)

Designations and Abbreviations

The dental record is an important medicolegal document. Not only does it facilitate diagnosis, treatment planning, and practice management, it is also a valuable means of communication between the primary clinician and other providers, and it may be used in defense of allegations of malpractice and aid in the identification of a dead or missing person. The record of the initial database shows missing teeth, existing restorations, and diseases and other abnormalities, while the chronological record of progress notes reflect treatment provided and diseases and other abnormalities that have occurred after the initial examination. The dental record is also a source of important information for the ongoing monitoring and evaluation of oral healthcare. Consequently, the charted record of the clinical process must be in conformity throughout the dental record.

While there are acceptable alternatives, for purposes of brevity and exactness, the alphabetical designation of primary teeth (Table 1.9) and the numerical designation of permanent teeth are advocated (Table 1.10).

Table 1.9. Alphabetical designation of primary teeth.

Tooth	Designation
Right maxillary primary second molar	A
Right maxillary primary first molar	B
Right maxillary primary cuspid	C
Right maxillary primary lateral incisor	D
Right maxillary primary central incisor	E
Left maxillary primary central incisor	F
Left maxillary primary lateral incisor	G
Left maxillary primary cuspid	H
Left maxillary primary first molar	I
Left maxillary primary second molar	J
Left mandibular primary second molar	K
Left mandibular primary first molar	L
Left mandibular primary cuspid	M
Left mandibular primary lateral incisor	N
Left mandibular primary central incisor	O
Right mandibular primary central incisor	P
Right mandibular primary lateral incisor	Q
Right mandibular primary cuspid	R
Right mandibular primary first molar	S
Right mandibular primary second molar	T

Table 1.10. Numerical designation of permanent teeth.

Tooth	Designation
Right maxillary third molar	1
Right maxillary second molar	2
Right maxillary first molar	3
Right maxillary second bicuspid	4
Right maxillary first bicuspid	5
Right maxillary cuspid	6
Right maxillary lateral incisor	7
Right maxillary central incisor	8
Left maxillary central incisor	9
Left maxillary lateral incisor	10
Left maxillary cuspid	11
Left maxillary first bicuspid	12
Left maxillary second bicuspid	13
Left maxillary first molar	14
Left maxillary second molar	15
Left maxillary third molar	16
Left mandibular third molar	17
Left mandibular second molar	18
Left mandibular first molar	19
Left mandibular second bicuspid	20
Left mandibular first bicuspid	21
Left mandibular cuspid	22
Left mandibular lateral incisor	23
Left mandibular central incisor	24
Right mandibular central incisor	25
Right mandibular lateral incisor	26
Right mandibular cuspid	27
Right mandibular first bicuspid	28
Right mandibular second bicuspid	29
Right mandibular first molar	30
Right mandibular second molar	31
Right mandibular third molar	32

To record pathologic conditions and subsequent restorations of teeth, the following designations of tooth surfaces are used universally: facial (F), lingual (L), occlusal (O), mesial (M), distal (D), and incisal (I). Clinical circumstances may require the use of combinations of designations to identify and locate caries and to record treatment plans, operations, or restorations in the teeth involved. For example, 8-MID would refer to the mesial, incisal, and distal aspects of a right maxillary central incisor; 22-DF, the distal and facial aspects of a left mandibular cuspid; and 30-MODF, the mesial, occlusal, distal, and facial aspects of a right mandibular first molar.

When charting missing teeth, existing restorations, and prostheses as part of initial documentation of the database (Table 1.11);

Table 1.11. Standardized chart markings for missing teeth, existing restorations, and prostheses.

Missing teeth	Draw a large "X" on the root or roots of missing teeth.
Edentulous mouth	Inscribe crossing lines, one extending from the maxillary right third molar area to the mandibular left third molar area and the other from the maxillary left third molar area to the mandibular right third molar area.
Edentulous arch	Inscribe crossing lines, each running from the uppermost aspect of the third molar area to the lowermost aspect of the third molar area on the opposite side.
Amalgam restoration	In the diagram of the tooth, draw an outline of the restoration showing size, location, and shape, and block in solidly.
Nonmetallic permanent restoration	In the diagram of the tooth, draw an outline of the restoration showing size, location, and shape.
Gold or other alloy restoration	In the diagram of the tooth, draw an outline of the restoration showing size, location, and shape, and inscribe horizontal lines within the outline. If made of an alloy other than gold, indicate in the REMARKS section that the restoration is made of a metal other than gold (where possible, indicate type of alloy used).
Combination restoration	In the outline of the tooth, draw an outline of the restoration showing size, location, and shape; and partition at junction of materials used and indicate each as above.
Porcelain or acrylic facings and pontic	In the diagram of the tooth, draw an outline of the restoration. Indicate in the REMARKS section that the facing or pontic is made of porcelain or acrylic.
Porcelain or acrylic post crown	In the diagram of the tooth, draw an outline of the restoration; outline approximate size and position of the post or posts. Indicate in the REMARKS section that the crown is made of porcelain or acrylic.
Porcelain or acrylic crown	In the diagram of the tooth, draw an outline of the restoration. Indicate in the REMARKS section that the crown is made of porcelain or acrylic.
Fixed partial denture	In the diagram of each tooth, draw an outline of the restoration; partition at junction of materials used. If made of gold, inscribe diagonal lines for both abutments and pontics. If made of an alloy other than gold, indicate in the REMARKS section that the restoration is made of a metal other than gold (where possible, indicate type of alloy used). Facing material should be indicated in the REMARKS section.
Removable prosthesis	Place a line over numbers of replaced teeth and describe briefly in REMARKS.
Root canal fillings	Outline each canal filled on the diagram of the root or roots of the tooth involved and block in solidly.
Apicoectomy	Draw a small triangle on the root of the tooth involved, apex away from the crown, the base line to show the approximate level of the root amputation.
Temporary restoration	In the diagram of the tooth, draw an outline of the restoration showing size, location, and shape. If possible, describe the material in REMARKS.

when charting diseases and abnormalities (Table 1.12); or when charting treatment completed (Table 1.11), standardized chart markings will further facilitate efficient continuity of care and may establish forensic identification.

Finally, when writing progress notes, the use of standard abbreviations and acronyms may be desirable for expediency (Table 1.13). In addition, the use of well-known medical and scientific signs and symbols, such as Rx, WNL, BP, H_2O, and others, is recommended.

Table 1.12. Standardized chart markings for diseases and abnormalities.

Caries	In the diagram of the tooth, draw an outline of the carious portion, showing size, location, and shape, and block in solidly.
Defective restorations	In the diagram of the tooth, outline the defective restoration and block in solidly.
Fractured tooth	Indicate approximate location of fracture with a zigzag line on outline of the tooth.
Partially erupted tooth	In the diagram of the tooth, draw an arcing line through the long axis.
Drifted teeth	Draw an arrow at the designating number of the tooth that has moved, with the point of the arrow indicating the direction of movement. Describe briefly in REMARKS.
Impacted tooth	Outline all aspects of each impacted tooth with a single oval. The long axis of the tooth should be indicated by an arrow pointing in the direction of the crown.
Radiolucency	Outline approximate size, form, and location.
Radiopacity	Outline approximate size, form, and location, and block solidly.
Periodontal status	PSR scores (PSR periodontal probe with a 3.5 mm ball tip and a 3.5–5.5 mm color-coded area) 0: Colored area of the probe remains completely visible in the deepest probing depth in the sextant. No calculus or defective margins are detected. Gingival tissues are healthy and no bleeding occurs after gentle probing. 1: Colored area of the probe remains completely visible in the deepest probing depth in the sextant. No calculus or defective margins are detected. There is bleeding after gentle probing. 2: Colored area of probe remains completely visible in the deepest probing depth in the sextant. Supra- or subgingival calculus or defective margins are detected. 3: Colored area of probe is only partly visible in the deepest probing depth in the sextant. 4: Colored area of probe completely disappears, indicating a probing depth of greater than 5.5 mm.

Table 1.13. Standard abbreviations and acronyms.

Acute necrotizing ulcerative gingivitis	ANUG	Oral health counseling	OHC
All caries not removed	ACNR	Oral surgery	OS
All caries removed	ACR	Panoramic radiograph	Pano.
Amalgam	Am.	Patient	Pt.
Anesthetic(thesia)	Anes.	Patient informed of examination findings and treatment plan	PTINF
Assessment	A	Periapical	PA
Camphorated paramonochlorophenol	CMCP	Pericoronitis	PCOR
Chief complaint	CC	Periodontal screening and recording	PSR
Complete denture	CD	Periodontics	Perio.
Copal varnish	Cop.	Plan	P
Crown	Cr.	Plaque control instructions	PCI
Curettage	Cur.	Porcelain	Porc.
Drain	Drn.	Postoperative treatment	POT
Electric pulp test	EPT	Preparation	Prep.
Endodontics	Endo.	Preventive dentistry	PD
Equilibrate(ation)	Equil.	Prophylaxis	Pro.
Eugenol	Eug.	Prosthodontics	Pros.
Examination	Exam.	Removable partial dentures	RPD
Extration(ed)	Ext.	Restoration(s)	Rest.
Fixed partial denture	FPD	Return to clinic	RTC
Fluoride	Fl.	Root canal filling	RCF
Fracture	Fx.	Root canal therapy	RCT
Gutta percha	GP	Rubber dam	RD
Health questionnaire reviewed	HQR	Scaled(ing)	Scl.
History	Hx.	Subjective	S
Mandibular	Man.	Surgical(ery)	Surg.
Maxillary	Max.	Suture(s)(d)	Su.
No significant findings	NSF	Temporary	Temp.
Objective	O	Topical	Top.
Operative	Oper.	Treatment(ed)	Tx.
Oral cancer screening exam	OCSE	Zinc oxide and eugenol	ZOE
Oral diagnosis	OD		

Conclusion

It is axiomatic that in the clinical process the primary customer is the patient. However, the customer may also be a member of one's own organization (associates, staff) or individuals/organizations outside the institution (consultants, insurance companies, lawyers) who are "downstream" in the clinical process and must work with the product that is handed down to them. The licensed dental practitioner is solely responsibly for all patient care–related activities including those legally provided by auxiliary personnel. This includes obtaining and documenting the patient's history, performing the physical examination, establishing diagnoses, developing and implementing preventive and therapeutic strategies, and properly documenting all services rendered and pertinent communications with patients.

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2 The Historical Profile

"Never treat a stranger." Sir William Osler's statement is especially applicable to the practice of dentistry, in which the physical and emotional ability of the patient to undergo and respond to dental care is determined primarily by reviewing the medical/dental history. An initial historical profile (Table 1.5) should identify the patient; establish the chief complaint; reflect the dental history; document drug allergies or other adverse drug effects; identify medications, vitamins, dietary supplements, or special diets; and provide a record of past and present illness, major hospitalizations, and a review of major organ systems. The historical profile shall be reviewed with the patient at each subsequent appointment and any new information obtained should be documented in the progress notes (Table 1.7).

The current trend among dental practitioners is to use a combined printed and oral approach to establish the historical profile of a patient. A written questionnaire will elicit information that may be omitted by oral inquiry. Oral communication will provide important insight into a patient's feelings about past, present, and future illnesses and courses of treatment. This process is critical to the patient-doctor relationship and establishment of the rapport that precedes successful treatment.

Clinicians must be aware of the patient's overt and hidden concerns and develop a sense of the patient's reliability as an interpreter and reporter of events. Patients may suppress some information purposely or unknowingly. They may under-report other experiences or present them in a context that is less disconcerting than might be appropriate. Circumstances that may be of concern to clinicians might not be seen as unusual to patients. Practitioners must be compulsive in compiling data, directing careful attention to the obvious and maintaining sensitivity to the less obvious "soft" clues that may be revealed in the history. An appreciation of the patient's perspective and an attitude of friendliness and respect will go a long way in assuring the patient's cooperation in gathering information.

Failure to obtain an initial historical profile, or to update it regularly, is not an excuse for being unaware of a patient's physical and emotional problems. Responses should be explored to determine whether the patient understands the question, is certain

of the answer, and appreciates the importance of the question and the answer in the context of the care to be provided. If the patient is confused, the dentist has an obligation to educate the patient to respond to questions, or the dentist may need to seek the necessary information from an additional informant. In all cases, the dentist should reduce those responses to writing. Failure to document and correctly interpret the historical profile of a patient may have devastating effects for the patient and the clinician.

Patient Identification

The basic biographical data should include the patient's name, age, sex, ethnic extraction, marital status, occupation, and place of residence. The date of the evaluation also must be recorded. Not only are these items essential for patient identification but also they may provide invaluable background information for the differential diagnosis of certain conditions, or identify patients in a high-risk category for a variety of diseases. For example, healthcare workers, military personnel, immigrants from developing countries, and people who work or live in institutions should be considered at higher risk for harboring certain infectious or communicable diseases.

Chief Complaint (Problem)

The dentist must record the patient's description of signs and symptoms associated with the current oral condition in a logical sequence. A clinician should begin with the chief complaint, stated in the patient's own words. An attempt should be made to determine why the patient is consulting the dentist today and not yesterday or tomorrow. The answer may reveal an important clue to the severity of pain, underlying emotional problems, or other matters that are important in the overall understanding of the patient's illness. Did acute symptoms prompt the visit, or was it the desire for a checkup because a neighbor, friend, or another family member was told they have oral cancer or have been diagnosed with HIV infection? The dentist should remember that a patient's expressed reason for seeking advice might mask underlying concerns. After an understandable statement of the chief complaint has been elicited, the chronology of the illness should be delineated.

Character of the Problem

The most common complaint causing a person to seek the services of a healthcare provider is pain. Determine its character. Is it sharp or dull? Is it pain or is it merely discomfort? Does it appear suddenly and disappear quickly, or does it gradually increase in intensity and subside slowly? A lesion should be inspected. Is it white, red, pigmented, ulcerative, vesicular, bullous, exophytic, or a combination of these various characteristics? Admittedly, this observation is part of the examination, not the history, but there are at least two good reasons for doing it at this point in time. First, it establishes the dentist's concern for the patient's problems, and second, it may suggest additional questions to be asked during the history-taking process.

Duration and Progression of the Problem

A number of questions should be considered. How long has the condition associated with the patient's chief complaint been present? Has the problem developed slowly or rapidly? Some conditions have a sudden onset, but others begin slowly and insidiously. Have the symptoms become worse or better? Are they better at times and worse at other times?

Domain of the Problem

One must determine whether the pain or discomfort remains localized or radiates to other anatomic locations. When dealing with a lesion, the dentist should determine if it is found on the lips, tongue, buccal mucosa, hard or soft palate, floor of the mouth, or other areas of the head and neck, because the location of a disease may assist in its diagnosis.

Relationship between Physiologic Function and the Problem

One should evaluate the effects of normal activities on the symptoms. What is the effect of the problem on mastication? Are the symptoms worse when the patient is chewing? In some instances, mastication relieves symptoms; in others, it aggravates them. Similar insights into the effects of swallowing, drinking, and speaking on the symptoms should be obtained.

Dental History

Important elements of a past dental history include frequency of visits to the dentist, history of radiographic examinations, type of care received in the past, history of orofacial injuries, and difficulties with past treatment. A history of adverse reactions to local anesthetic agents, latex products (gloves, rubber dam), or other dental materials should also be investigated. Note the attitude of the patient toward previous dentists and therapeutic interventions. Is this a patient who will never be satisfied no matter the skill of the clinician, or does the patient have significant undiagnosed problems that form the basis of the chief complaint? What is the patient's dental IQ and what priority is the patient likely to place on home care following periodontal surgery or extensive restorative care?

Medical History

The oral healthcare provider should document a history of allergic drug reactions and other adverse drug effects and investigate whether drugs or medications are being taken. Many patients habitually take drugs for minor complaints, a practice that should be documented carefully. Patients often do not recognize nonprescription medications as drugs and, therefore, do not mention the habitual use of aspirin, decongestants, antihistamines, vitamins, and many other over-the-counter medications. Inquire about dietary supplements or special diets the patient may be on. Immunosuppressant therapy may place a patient in the high-risk category for many viral, fungal, and bacterial infections and de novo malignancies.

The dentist should inquire about the patient's general health, as perceived by the patient, and summarize past and present medical conditions. A clinician must record any hereditary or developmental abnormalities. Patients with hemophilia or other coagulopathies are at a higher risk for hepatitis and HIV infection because of the potential for undergoing multiple blood transfusions. Previous operations, injuries, accidents, and hospitalizations should be recorded, as well as comments about anesthesia, drug reactions, blood transfusions, or transmissible diseases. A history of repeated hospitalizations for the same condition, failure of an infection to resolve following therapy, recurrent infections with the same pathogen, and infection with unusual organisms, especially in the absence of "hard" signs of infection, may be suggestive of hereditary or acquired immunodeficiency, or therapeutic immunosuppression.

Family History

Diabetes mellitus, hypertension, dyslipidemia, and allergic reactions have a signifi-

cant tendency to appear in certain families, and certain types of cancer stalk through generations of the same family. The family history is particularly important in assessing diseases of the nervous system. Some conditions such as hemophilia, passed on by an affected mother to her sons, are always hereditary. In addition to hereditary conditions, acquired infectious diseases may be transmitted from one family member to another, some requiring only casual contact, while others are transmitted only through repeated, intimate encounters (sometimes associated with child abuse).

Because of the frequency of facial and intraoral injuries and/or the presence of suspected sexually transmitted diseases associated with family violence (child abuse, spouse abuse), the oral healthcare provider is likely to be the first professional to observe the victim. While obtaining the history of the problem, careful attention must be paid to the explanation provided by the patient or other family members relative to the suspected problem. Look for any inconsistencies or behaviors suggestive of reluctance to provide information. Note nonverbal behaviors, which may not match verbal statements. The dentist does not have to determine abuse. However, reasonable suspicions should be reported to the appropriate local or state agency for follow-up.

Social History

The personal habits of patients may reveal important clues to diagnosis. Excessive use of tobacco and alcohol may produce symptoms whose significance is lost without knowledge of a patient's smoking and drinking habits. The daily use of tobacco products should be recorded in numbers of cigars, cigarettes (packs), or pipefuls smoked. Alcohol use is unequivocally associated with child abuse, fatal traffic accidents, homicides, rapes, and suicides. Alcohol consumption should be recorded in terms of quantity and type over a specific period of time. Since patients with alcoholism are especially prone to certain diseases, it is important not to overlook this particular finding. The simple question "when was the last time you had more than X drinks in 1 day?" where X equals five for men and four for women should be asked as part of the interview. A response of "within the past 3 months" usually indicates the patient has a drinking problem and should undergo further assessment.

The patient's social history may also alert the clinician to the presence of environmental and cultural factors that may significantly influence the patient's general health and provide insight into the patient's personality and emotional state. A history of recreational drug use, frequent moves, sexual promiscuity (whether homosexual, bisexual, or heterosexual), frequent travels to developing countries, or recent immigration into the United States should alert clinicians to patients at high risk for infectious diseases. Information about educational, social, religious, and economic background and feelings of achievement or frustration can provide important insight into understanding the patient as a person. From this information, one can assess which factors might have a bearing on the current problem and whether they might be supportive or stressful influences.

Review of Organ Systems

The chief complaint and the medical, family, and social histories of the patient should guide the clinician to investigate areas of special concern. All signs and symptoms related to specific organ systems should be recorded. The status of organ systems may suggest the presence of concomitant systemic conditions, contribute to the diagnostic process, and influence projected treatment protocols and prognosis.

Skin

Itching, Rash, and Ulcers

An important cause of pruritus, especially associated with a bitter metallic taste and burning tongue, may be psychogenic (e.g., a reaction to stress and strain). A subtle and important cause of pruritus without a visible rash may be a reaction to drugs, such as aspirin, opiates and their derivatives, heroin, or amphetamine abuse. Generalized pruritus is frequently the first sign of biliary cirrhosis and may occur many months before the onset of jaundice. It may also be associated with carcinoma or a hematological disorder such as polycythemia vera, Hodgkin's lymphoma, or T-cell lymphoma (Sezary syndrome). Patients with pruritus in association with obvious skin lesions, such as papules, vesicles, bullae, or ulcerations, should be referred to a dermatologist. Many of these disorders require specialized dermatological approaches to establish the diagnosis.

Pigmentations

Vitiligo is an acquired depigmenting disorder characterized by localized or generalized hypomelanosis of the skin and hair. Its etiopathogenesis is poorly understood, but likely involves multiple overlapping pathogenic mechanisms. When localized, hypomelanosis of the skin and hair may be restricted to one region, such as the scalp. When generalized, the pattern of hypomelanosis is quite typical, with lesions particularly on the face and neck coupled with loss of pigment in the hair.

Neurofibromatosis (von Recklinghausen disease) is inherited as an autosomal dominant trait. It is characterized by the appearance of numerous cutaneous café-au-lait spots. The majority of these lesions occur on the trunk and vary in diameter from less than 1 cm to more than 15 cm. The presence of six or more café-au-lait spots, each with a diameter greater than 1.5 cm (> 0.5 cm in children), is highly suggestive of neurofibromatosis even without a familial history of the disease.

Peutz-Jeghers syndrome is an autosomal dominant trait associated with intestinal polyposis and mucocutaneous pigmentation. The polyposis is most frequent in the ileum and jejunum and the mucocutaneous hypermelanosis is most noticeable in periorificial sites and the oral mucosa. It is now recognized that patients with Peutz-Jeghers syndrome are at increased risk for developing both gastrointestinal and nongastrointestinal malignancies.

Diffuse brown hypermelanosis is a striking feature of primary adrenocortical insufficiency (Addison's disease). Most cases are caused by an autoimmune process or infiltration of the gland by an infectious agent (HIV, MBT). There is significant accentuation of pigmentation in certain mucocutaneous areas, namely along pressure points and oral mucous membranes. These patients demonstrate hypotension and a decreased tolerance to stress associated with infection, surgery, or trauma. An identical type of diffuse hyperpigmentation also has been reported as a sequela of adrenalectomy in patients with Cushing's disease (Nelson syndrome). A third example of the Addisonian type of hypermelanosis has been reported in patients with pancreatic and lung tumors. This phenomenon is known as a paraneoplastic syndrome.

In certain chronic nutritional deficiencies, splotches of dirty-brown hyperpigmentation may appear, especially on the trunk. Patients with protein deficiency may demonstrate a change in hair color, first to reddish brown and eventually to gray. In other selective deficiencies, such as sprue (faulty absorption of fats and carbohydrates), the hypermelanosis may be distributed over any area of the body, whereas in pellagra (niacin deficiency), it is limited to skin that is exposed to light or irritation. In vitamin B_{12} deficiency, the hair loses it original color and becomes gray and

there is a diffuse cutaneous distribution of hypermelanosis.

Lack or Loss of Body Hair

Male-pattern baldness is inevitable in the presence of androgenic stimuli in patients with a genetic predisposition to baldness. The hypopituitary dwarf may completely lack hair, while patients with acquired hypopituitary states rapidly lose hair from the axillae, pubis, and, at times, the scalp. In congenital cretinism, lanugo hair may be retained, but the scalp hair is sparse and dry. In adults, hypothyroidism causes a decrease in secondary sexual or hormonal hair, in addition to the characteristic loss of the lateral third of the eyebrows. The loss of scalp hair in a male pattern along with an increase in body and facial hair may be due to increased production of adrenal androgens (Cushing's syndrome) or exogenous adrenocorticotropic hormone administration.

In women, postpartum increase in hair loss is normal. The prolonged growth phase resulting from hormonal stimulation during pregnancy ends after delivery, and a synchronized onset of the resting phase occurs in the scalp hair follicles. Prolonged febrile illnesses, systemic lupus erythematosus, dermatomyositis, severe cachexia, and lymphomas also may be associated with hair loss. Permanent hair loss on the extensor surfaces of the fingers is an early sign of systemic scleroderma. Superficial ringworm infections of the scalp, deep pyogenic infections, and severe herpes zoster are associated with permanent hair loss in the affected area. Permanent alopecia may occur in lesions of discoid lupus erythematosus, localized scleroderma, and sarcoidosis, usually involving the scalp and eyebrows. Ionizing radiation in large doses causes permanent hair loss. Transient hair loss may be caused by certain medications such as antimetabolites, heparin, coumarin, and excessive doses of vitamin A.

Extremities

Varicose Veins

Varicosities may occur secondary to thrombophlebitis, trauma, increased venous pressure, pregnancy, or heart failure. Extensive varicose veins may lead to dizziness and syncope under conditions of orthostatic stress.

Swollen or Painful Joints

The causes of joint disorders are numerous and include traumatic, infectious, metabolic, immunologic, and neoplastic processes. Joint disorders may produce pain, stiffness, swelling, redness, increased warmth, or limitation of motion. Edema associated with heart failure tends to be most extensive in the ankles and accentuated in the evening, a feature determined largely by posture. Other evidence of heart disease usually indicates the pathogenesis of edema.

Muscle Weakness and Pain

Reduced strength of contraction, diminished power with single contractions, and repeated contractions are indubitable signs of muscle disease. In most of these diseases, some of the muscles are affected and others are spared. Each disease exhibits its own pattern. Ocular palsies are seen more or less exclusively as diplopia (double vision), ptosis (drooping eyelids), or strabismus (deviation of the eye that cannot be overcome by the patient). Facial palsy is seen as an inability to close the eyes or smile and expose the teeth. Bulbar palsy is seen as dysphonia, dysarthria, and dysphasia, with or without a hanging jaw or facial weakness. Cervical palsy is often seen as the hanging-head syndrome, which is defined as an inability to lift the head from a pillow.

Bone Deformities or Fractures

Bone is a dynamic tissue that is remodeling itself throughout life. The response of bone

to injuries, such as fracture, infection, interruption of blood supply, and the presence of expanding lesions, is relatively limited. Dead bone must be resorbed and new bone formed. Even in an architecturally disruptive disorder, remodeling appears to be dictated by mechanical forces. Disorders involving osseous tissues are associated with calcium, phosphorus, calcitonin, vitamin D, and parathyroid hormone interactions.

Prosthetic Joints

Of the many potential complications after total joint replacement, infection is by far one of the most serious. There have been several reports of infection in hip prostheses, apparently resulting from bacteria seeded from infections of the kidney, lungs, or gastrointestinal tract. The circumstantial association reported between certain dental procedures and transient bacteremias and the possibility of metastatic infection of artificial prostheses should be a point of concern. Patients at potential increased risk of hematogenous total joint infection, such as those with therapeutic or acquired immunodeficiency, rheumatoid arthritis, systemic lupus erythematosus, type I diabetes mellitus, previous prosthetic joint infection, hemophilia, malnutrition, and those within the first 2 years following joint replacement, should receive antimicrobial prophylaxis prior to procedures likely to cause bleeding. Antibiotic prophylaxis is not indicated for dental patients with pins, plates, or screws.

Eyes

Conjunctivitis

Conjunctivitis associated with burning, itching, and runny eyes might be apparent in patients with allergies, the common cold, herpes keratitis, or gonococcal or chlamydial infections. A history of icteric sclera combined with hemolytic or obstructive liver disease suggests hepatocellular jaundice possibly resulting from acute viral, drug-induced, or alcoholic hepatitis; subacute or chronic hepatitis; or cirrhosis.

Blurred Vision

The dentist should record whether the patient wears glasses or contact lenses. The appearance of black spots moving in front of the eyes, followed by nausea, is the first and most common sign of migraine headache. Blurred vision may also result from cataracts (often caused by diabetes mellitus), Stevens-Johnson syndrome, or cicatricial pemphigoid.

Double Vision

Diplopia occurs when the disparate points (visual receptors) are too far apart. The images formed are separate and do not fuse. Diplopia may occur when the area in the cerebrum for visual acuity is compromised by trauma, stroke, or vascular abnormalities. It is also the predominant symptom of dysfunction of the optic nerve.

Drooping Eyelids

Paresis of the third cranial nerve (oculomotor) will result in ptosis. Drooping of the eyelid may also be an early sign of myasthenia gravis and Horner's syndrome (paralysis of the cervical sympathetic nerves characterized by ptosis, constriction of the pupil, anhydrosis, and flushing on the affected side of the face).

Glaucoma

Glaucoma is characterized by increased intraocular pressure associated with progressive irreversible damage to the optic nerve, resulting in defects in the visual field. It is the most common cause of blindness in many areas of the world. Acute primary open-angle glaucoma is associated with a sudden increase in intraocular pressure. The eye is immobile, the pupils are dilated, and the cornea is edematous. Severe aching and pain are present.

Chronic primary open-angle glaucoma represents the most common type of glaucoma.

Ears, Nose, and Throat

Earache and Tinnitus

Patients with a history of recurrent ear infections may exhibit pain referred to the dentition or temporomandibular joint, while pain of odontogenic or myofacial origin may mimic otitis media. Tinnitus, or ringing of the ears, is a purely subjective phenomenon. It is a common complaint in adults, but often of no clinical significance. A hissing sound may result from a build-up of wax in the external auditory canal or a blocked eustachian tube. It is commonly associated with arteriosclerosis.

Hearing Loss

The most common causes of middle ear deafness are otitis media, otosclerosis, and rupture of the eardrum. Nerve deafness has many causes, including damage from rubella or syphilis. The auditory nerve may be affected by tumors of the cerebellopontine angle. Deafness also may result from a demyelinating plaque in the brain stem. Fullness, vertigo, tinnitus, and fluctuating hearing loss may be due to Ménière's disease, a rare and poorly understood nonsuppurative disease of the labyrinth.

Nose Bleeds

The most common cause of epistaxis is probably nose picking, leading to tearing of the rich network of veins (Kiesselbach plexus) in the anterior naris. Minor epistaxis also may appear in the course of viral infections of the upper respiratory tract. Other causes of intermittent or repeated episodes of epistaxis are atheromas of the nasal vessels, hypertension, bleeding diatheses (thrombocytopenia, coagulopathies), polycythemia vera, rhinoliths, acute sinusitis (especially involving the ethmoid sinus), tumors of the nose and paranasal sinuses, nasal angiomas, and Wegener granulomatosis. The number of bleeding episodes along with the severity of epistaxis is frequently increased in patients taking antithrombotic agents or anticoagulants. In hereditary hemorrhagic telangiectasia, the nose may be the only site of bleeding.

Sinusitis

The most common predisposing factor for acute purulent sinusitis is a viral infection of the upper respiratory tract. This may lead to obstruction of the paranasal sinuses along with the development of localized pain, tenderness, and low-grade fever. Frontal sinusitis is characterized by pain over the forehead. Pain, swelling, and tenderness in the anterior portions of the maxilla characterize maxillary sinusitis. Ethmoid sinusitis is characterized by pain in the upper lateral areas of the nose, frontal headache, redness of the skin, and tenderness to pressure over the nasal bones adjacent to the inner canthus of the eye. Sphenoid sinusitis is characterized by tenderness and pain over the vertex of the skull, mastoid bones, and occipital portion of the head. These manifestations usually clear as the viral disease subsides. In a number of instances, however, invasion by pyogenic bacteria supervenes and causes a purulent sinusitis to develop. The cause of chronic sinusitis may be the same as that for the acute form, but more than one pathogen may be present. A neoplastic lesion should be ruled out in patients who experience repeated episodes of acute sinusitis or who have chronic symptoms.

Sore Throat

A sore throat, regardless of the cause, is the outstanding symptom of acute pharygnitis. Approximately two-thirds of all acute illnesses are viral infections of the upper respiratory tract that demonstrate varying degrees of pharyngeal discomfort. The most common complication of acute pharyngitis is

peritonsillar cellulitis and abscess. Pharyngitis also can be a symptom of an oropharyngeal gonococcal infection or, when associated with low-grade chronic fever and malaise, it may be the initial manifestation of hepatitis. Persistence of pain in an enlarged firm tonsil, in the absence of an infectious process, is an indication for biopsy. The presence of fever does not rule out a neoplastic lesion because the temperature may be elevated in lymphomas.

Hoarseness

Laryngitis is the most common symptom of a disorder involving the larynx and it often interferes with normal phonation. Although hoarseness is usually of short duration, with acute self-limited processes such as infections, it may persist for long periods and may be a common complication of gastroesophageal reflux disease. When hoarseness has persisted for longer than 2–3 weeks, the cause of laryngeal obstruction should be determined.

Respiratory Tract

Shortness of Breath

Dyspnea, difficult or labored breathing, is associated with abnormalities resulting in hypoxia, or even more commonly with disorders associated with excess carbon dioxide retention. It is a cardinal manifestation of diseases involving the respiratory and cardiovascular systems. Dyspnea that is present at rest or when performing a menial task is an early manifestation of left ventricular heart failure. Orthopnea and acute paroxysmal nocturnal dyspnea may also be present. The dyspnea of chronic obstructive pulmonary disease tends (COPD) to develop more gradually than that of heart disease.

Coughing

Cough is one of the most frequent respiratory symptoms produced by inflammatory, mechanical, chemical, and thermal stimulation of the cough receptors. It is an explosive expiration that helps clear the tracheobronchial tree of secretions and foreign bodies. Acute episodes of cough may be associated with viral infections such as acute tracheobronchitis or pneumonitis or with bacterial bronchopneumonia. Chronic cough is a common annoyance that causes anxiety, urinary incontinence, insomnia, and exhaustion. In addition to smokers and others exposed to environmental irritants, patients with a chronic cough often suffer from postnasal drip syndrome, gastroesophageal reflux disease, or left-ventricular failure. They may also be taking angiotensin-converting enzyme inhibitors. Coughing is so common in cigarette smokers that it is often ignored or minimized. Any change in the nature and character of a chronic cough by a cigarette smoker should prompt an immediate diagnostic evaluation, with particular attention directed to the detection of pulmonary tuberculosis and bronchogenic carcinoma.

Hemoptysis

Hemoptysis, or blood in the sputum, may be evidence of a respiratory tract infection or a pulmonary neoplasm. A productive cough in the morning characterized by hemoptysis is highly suggestive of tuberculosis, especially if associated with pain, dysphagia, dysphonia, and significant weight loss. Although hemoptysis may occur during the course of a viral or bacterial pneumonia, its occurrence always should raise the question of a more serious underlying process.

Bronchitis and Emphysema

Bronchitis, or chronic inflammation of the bronchi and bronchioles, is most commonly observed in smokers. These patients are usually heavyset, blue or red-blue around the face, and have distended neck veins and ankle edema. They may be taking bronchodilators and experience frequent pulmonary infections.

Emphysema usually is preceded by chronic bronchitis and is characterized by irreversible obstructive disease with dilation and destruction of the acinar walls. The patients are generally thin, pink, carry their shoulders high, and breathe with their intercostal muscles. Oxygen must be used with care in patients with chronic obstructive pulmonary disease (chronic bronchitis and emphysema) because the respiratory center in the brain readjusts so that the basic stimulus to respiration is oxygen instead of carbon dioxide.

Wheezing and Asthma

Wheezing is a whistling sound made during expiration and is usually seen in association with asthma. Bronchial asthma is a respiratory disease characterized by inflammation of alveolar epithelium, hypersecretion of mucus, and bronchial smooth muscle spasm presenting as the triad of coughing, wheezing, and labored breathing (dyspnea). Allergens, upper respiratory tract infections, exercise, nonsteroidal anti-inflammatory agents, and emotional stress may provoke an asthmatic attack. The association of aspirin-induced asthma, aspirin sensitivity, and nasal polyps is known as Samter triad. Dental treatment may also trigger a reaction in the hyperactive airways. A clinically significant decrease in lung function has been reported in up to 15% of children with asthma. Wheezing is regarded as the sine qua non. In its most typical form, asthma is an episodic disease, and all three symptoms coexist.

Latent TB Infection and TB Disease

Ninety to 95% of the infections with *Mycobacterium tuberculosis* (MBT) are subclinical, producing only a positive tuberculin skin test and a latent tuberculosis infection (LTBI). LTBI may become active and produce active TB disease. The risk of active TB disease is greatest in the first 2 years after initial infection and it is estimated that one in ten persons with LTBI will develop active TB disease unless preventive therapy is initiated. The onset of active TB disease in susceptible patients may be delayed for years or even decades and is often triggered in later life by medical conditions that alter the ability of the immune system to maintain the isolation in a latent state.

The lung is the most common target for active TB disease. It is characterized by a productive, prolonged cough (more than 3 weeks in duration); fever, chills, and night sweats; loss of appetite, weight loss, and easy fatigability; and hemoptysis. Active TB disease may be asymptomatic in its early stages and approximately 5% of all cases are reported initially at autopsy. About 15% of patients with active TB disease present with disease at an extrapulmonary site, which is most common in patients infected with HIV. The drug history can be quite useful in differentiating between those patients with a history of LTBI and those with a history of active TB disease.

Cardiovascular System

High Blood Pressure

Arterial pressure must be maintained at levels sufficient to permit adequate perfusion of the extensive capillary networks in the systemic vascular bed. A sustained elevation of arterial pressure results in secondary organ damage (i.e., cardiac, renal, or cerebrovascular effects). If this condition is unaltered by therapy, it may result in symptomatic illness and death. The goal in the management of hypertension is to reduce morbidity and mortality by lifestyle modification and pharmacotherapy. This may be accomplished by achieving and maintaining systolic blood pressure below 140 mm Hg and diastolic blood pressure below 90 mm Hg, while also controlling other modifiable risk factors for cardiovascular disease. Treatment to lower blood pressure is essential in order to prevent stroke, preserve renal function, and prevent or slow the progression of heart failure.

Chest Pain and Myocardial Infarction

Pain and pressure in the chest (angina pectoris) occur when the oxygen supply to the myocardium is deficient. By far the most frequent underlying cause is organic narrowing of the coronary arteries secondary to atherosclerosis. Although approximately 25% of myocardial infarctions (MIs) are silent, pain is the most frequent presenting complaint. The pain associated with MI is a deep visceral pain, similar in distribution to anginal pain, and the adjectives commonly used to describe it are heavy, squeezing, and crushing.

Congenital Heart Disease

Cardiac murmurs or abnormal heart sounds result from vibrations in the heart as a result of turbulent blood flow. Some murmurs are functional and of no consequence. However, murmurs associated with congenital or acquired heart disease often place the patient at a significantly increased risk for developing infective endocarditis (IE). Patients with cardiac conditions associated with the highest risk of adverse outcome from IE should be given prophylactic antibiotics before all dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa. Therefore, all patients with an equivocal history of congenital heart disease or previous IE should have a medical consultation prior to the initiation of dental care.

Prosthetic Valves and Pacemakers

Patients with prosthetic heart valves are at the highest risk of adverse outcome from IE and require antimicrobial prophylaxis before all dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa.

Cardiac pacemakers (intravascular or epicardial) or implanted cardiac defibrillators are often placed to override potentially life-threatening cardiac conduction abnormalities that may develop as a consequence of atherosclerotic heart disease, myocardial infarction, or heart block. For these patients, antibacterial prophylaxis is not recommended because the risk of developing IE is negligible (risk no greater than that in the general population).

Patients who have implanted pacemakers or ICDs may be susceptible to electromagnetic interference from outside sources that may result from electrical current generated by dental devices. Inhibition of pacing has been noted with electrosurgery units, ultrasonic scalers, and ultrasonic cleaners. The rate and rhythm of pacing appear not to be affected by dental handpieces, amalgamators, dental units and lights, endodontic ultrasonic instruments, sonic scalers, and radiographic units.

Gastrointestinal Tract

Eating Disorders

Difficulty swallowing is manifested as a sensation of food sticking somewhere in the esophagus. It may result from an emotional disturbance (globus hystericus), obstruction in the esophageal tract, muscular dysfunction, or local irritation. Bulimia nervosa, a condition observed mostly in young women, has a psychological basis. These patients can regurgitate at will, and as a consequence experience frequent episodes of acid reflux into the oral cavity. This in turn might result in chemical erosion of teeth in a peculiar pattern. Patients with suspected eating disorders should be referred for medical evaluation.

Gastroesophageal Reflux Disease (GERD)

The primary determinant of GERD appears to be a transient relaxation of the lower esophageal sphincter not induced by swallowing. Episodes of transient relaxation are more common after meals, in association with slow gastric emptying, and in the pres-

ence of increased intra-abdominal pressure. Transient relaxation of the lower esophageal sphincter is more likely to be followed by an episode of reflux when there is a hiatal pouch (hernia) containing retained gastric acid. GERD is further exacerbated by obesity and smoking (nicotine relaxes the lower esophageal sphincter). The squamous epithelium of the esophagus is not designed to resist the digestive action of gastric juice and it frequently becomes inflamed and eroded in patients with GERD. Complications of GERD include peptic strictures due to scarring of the inflammed tissue. Additionally, persistent reflux disease may lead to metaplastic tranformation of the esophageal squamous epithelium (Barrett's esophagus) and an increased risk of esophageal carcinoma.

Abdominal Pain and Peptic Ulcer Disease

Patients with abdominal pain relate classic patterns of pain distribution. The pain of a duodenal ulcer is localized to the right side of the abdomen, with a focal spot of tenderness, and a characteristic cycle of pain-food-pain relief-pain. Pain in the right upper quadrant may be a sign of liver disease, such as hepatitis; gallbladder dysfunction (cholecystitis); or carcinoma at the head of the pancreas. Pain in the right lower quadrant may result from acute appendicitis (McBurney point) or pneumonia in the superior aspect of the lower right lung lobe (fever with an elevated white blood cell count). Gastric ulcers produce a diffuse pain on the left side. Pain in the left upper quadrant may result from an enlarged spleen or pancreatic involvement (carcinoma or inflammation of the tail of the pancreas). Pain in the left lower quadrant may be a sign of diverticulitis. Pain in the epigastric area is indicative of acute appendicitis. In a diabetic patient, vague, diffuse abdominal pain associated with hyperventilation is a possible sign of a diabetic coma. All patients with abdominal pain require early and thorough evaluation because proper therapy often requires urgent action.

Liver Disease

The liver, which is the largest organ in the body, is involved in the formation of bile, urea, plasma albumin, fibrinogen, prothrombin, heparin, glycogen, and ketones. In addition, the detoxification of drugs and toxins, deamination of proteins, storage of glycogen, and excretion of drugs also occur in the liver. Hepatic structural and functional abnormalities occur in chronic cirrhosis, hepatitis, and a variety of pathophysiologic states, including congestive heart failure and metabolic, inflammatory, toxic, infectious, and neoplastic diseases. Disease-induced morphologic and functional changes of the liver can adversely affect hemostasis, alter drug metabolism, and impair the immune response to infections.

Jaundice

Yellow discoloration of the skin, mucous membranes, and sclera may results from increased concentrations of bilirubin in the blood. Bilirubin is a by-product of hemoglobin catabolism when red blood cells are destroyed in the spleen. This water-insoluble molecule is attached to albumin and is conjugated with glucuronic acid in the liver, rendering it water-soluble. The conjugated bilirubin becomes a constituent of the bile and is transported to the duodenum, where it gives the fecal matter its characteristic color. The predominance of conjugated or unconjugated bilirubin can pinpoint the metabolic problem as prehepatic (excess destruction of red blood cells), hepatic (hepatitis, cirrhosis, infectious mononucleosis, liver carcinoma), or posthepatic (cholelithiasis, carcinoma of the head of the pancreas).

Hepatitis

The major causes of hepatitis are alcohol, drugs, and viruses. Although viral hepatitis leads to acute hepatic failure in only a small number of patients (< 1%), the foremost

cause of acute liver failure is acute viral hepatitis, accounting for up to 72% of all cases. Approximately 80% of viral hepatitis infections are caused by the hepatotropic viruses A (HAV), B (HBV), C (HCV), D (HDV), or E (HEV). There are a number of other hepatotropic viruses; however, their contribution to the overall burden of hepatitis remains unknown. In addition, other viral agents can secondarily infect the liver, causing hepatitis; the most important of these are the Epstein-Barr virus, cytomegalovirus, and herpesviruses 1, 2, and 6. Liver disease may also occur following infections with rubella, rubeola, coxsackie, varicella-zoster, and adenoviruses. As a consequence of their parenteral mode of transmission and ability to establish chronic infection, hepatitis types HBV, HDV, and HCV are of particular concern for the oral heathcare professional. Hepatitis lasting for 6 months or more is generally defined as chronic and is classified according to the etiology and modified by the histologic status of the liver.

Genitourinary Tract

Difficulty Urinating (Dysuria)

Difficulty with or pain on urination may result from a wide variety of pathologic conditions. Evaluation of the condition must include a complete history and physical examination, as well as a complete urologic examination by an appropriate specialist. Dysuria may be associated with prostatic enlargement (hypertrophy or carcinoma), poststreptoccoccal glomerulonephritis, cystitis, urethritis, pyelonephritis, and gonorrhea.

Excessive Urination (Polyuria)

For practical purposes, there are four important disorders in which polyuria may occur. These include diabetes mellitus, diabetes insipidus, nephrogenic diabetes insipidus and acquired renal lesions, and psychogenic polydipsia.

Blood in Urine (Hematuria)

Bleeding from the urinary tract, whether microscopic or gross, is a serious sign, and must be regarded with the same gravity as abnormal bleeding from any other body orifice. The most common cause of hematuria is acute cystitis, but it may also be caused by hypertension with secondary renal damage, acute glomerulonephritis, trauma, a toxic response to drugs such as acetylsalicylic acid or acetaminophen, bladder carcinoma, and gonorrhea.

Kidney Disease

Bacterial infections of the urinary tract are extremely common. Many resist treatment and are likely to recur. Lower urinary tract infections are known as cystitis and urethritis, and the most common causative organisms are colonic flora and gonococci. Upper urinary tract infections are known as pyelitis and pyelonephritis. Chronic renal insufficiency, chronic renal failure, and end-stage renal disease are a continuum and may come to clinical attention because of the presence of anemia, hypertension, uremia, or growth retardation. The presence of renal disease affects both pharmacodynamic and pharmacokinetic mechanisms and may lead to significant drug toxicity.

Sexually Transmitted Diseases

Many sexually transmitted diseases have reached epidemic proportions. Syphilis, gonorrhea, chlamydia, herpes, condyloma acuminata, and HIV infection have significant oral manifestations. A correct diagnosis requires a through review of a patient's medical and social histories, as well as an understanding of the associated oral manifestations.

Endocrine System

Thyroid Abnormalities

An obese patient who demonstrates symptoms of fatigue, drowsiness, cold intolerance, and poor memory and has physical signs that include dry, coarse skin and hair, a decreased heart rate, and slow reflexes may have hypothyroidism. Conversely, a patient who relates symptoms of headache, increased appetite, weight loss, and heat intolerance and has physical signs that include exophthalmia, increased heart rate, and agitation may have hyperthyroidism.

Weight Change

Causes of weight gain may include overeating, hypothyroidism, edema with congestive heart failure, hepatic and renal failure, and Cushing's syndrome. Weight loss may be associated with diet, gastrointestinal dysfunction (peptic ulcer, gallbladder disease, enteritis, colitis), hyperthyroidism, malignancy, affective disorders (anxiety, depression, hysteria), adrenal insufficiency, and infection (HIV, tuberculosis, syphilis). Clinicians must take precautions when treating patients who have recently noticed a significant change in weight or who are grossly overweight or underweight because these patients may have altered sensitivity to depressants, sedatives, or analgesics.

Diabetes Mellitus

Diabetes mellitus is the most prevalent of all endocrine dysfunctions. From a clinical standpoint, concerns include insulin shock, diabetic coma, and a patient's inability to limit infection. As the disease progresses in severity, with glucosuria and ketonuria, weight loss occurs despite an increased appetite. Patients with Type I diabetes mellitus are usually younger, thinner, and ketosis prone, whereas patients with Type II diabetes mellitus tend to be older, obese, and generally ketosis resistant. Patients with Type I diabetes mellitus often exhibit polyphagia, polyuria, polydipsia, pruritus, and polyneuritis. Patients with Type II diabetes mellitus will exhibit more subtle symptoms, such as eye problems, ulcers on the legs and feet that do not heal properly, increased blood pressure resulting from atherosclerosis, renal dysfunction, and nonspecific oral complications.

Polydipsia

Excessive thirst usually suggests the possibility of diabetes mellitus or diabetes insipidus. This complaint may also occur as a result of severe hypokalemia (primary hyperaldosteronism, Cushing's syndrome, and excessive diuretic therapy).

Hematopoietic Abnormalities

Excessive Bleeding

Excessive bleeding is one of the most serious and cardinal manifestations of disease. It may occur in an isolated area or be more generalized in distribution. Bleeding associated with a localized lesion may be superimposed on a normal or defective hemostatic mechanism. In contrast, generalized bleeding usually is associated with a bleeding diathesis. In evaluating localized bleeding, the site of involvement, appearance of the blood, signs of blood loss, and evidence of disordered hemostatis should be considered. Excessive bleeding may be induced pharmacologically by antithrombotic agents, sodium warfarin, and heparin; or it may result from a defect in the hemostatic system.

Anemia

Anemia should never be thought of as a diagnosis in and of itself, but rather as a manifestation of an underlying disease process. In severe anemia, cardiac output increases. The patient may be aware of this increased cardiac activity and complain of palpitations. On examination, tachycardia and an increased

pulse pressure may be found. The adequacy of the cardiovascular adjustments to anemia depends on the degree of the anemia, the rapidity with which it has developed, and the pre-existing status of the cardiovascular system.

Pallor, headache, vertigo, faintness, increased sensitivity to cold, tinnitus or roaring in the ears, black spots before the eyes, muscular weakness, easy fatigability, and irritability are common neuromuscular symptoms associated with anemia. Loss of appetite is not an unusual complaint. Nausea, abdominal discomfort, constipation, diarrhea, and vomiting may also be associated with anemia. Menstrual disturbances and a loss of libido (in men) are encountered frequently in patients with severe anemia. Slight proteinuria and evidence of distinct impairment in renal function are also common genitourinary symptoms associated with anemia.

Abnormal White Blood Cell Count or Function

Alterations in leukocyte count and function occur in a wide variety of hematological, infectious, inflammatory, metabolic, and neoplastic diseases. Infections are a major cause of morbidity and mortality during periods of granulocytopenia. These infections are often associated with cancer chemotherapy, acute leukemia, aplastic anemia, and immunosuppression. The risk of infection is greatest when the polymorphonuclear leukocyte count falls below 500 per mm^3. When the granulocyte count is less than 100 per mm^3 for more than a few days, bacteremia and severe infection are inevitable.

HIV Infection and AIDS

Considering the incidence of HIV infection in the general population, dentists can expect to encounter infected individuals seeking care. Some of those patients will have AIDS or demonstrate symptoms associated with HIV infection, but many will be asymptomatic and thus may be unaware that they harbor the virus. Particular attention should be given to detecting oral and head and neck manifestations associated with the infection. The examination should take into consideration the patient's immunologic, hematologic, and general medical condition. The oral cavity is not considered to be a well-recognized site for the transmission of HIV (with the exception of breast-feeding and, possibly, oro-genital activity). Nonetheless, the oral mucosal defense mechanisms are not impermeable, particularly if the integrity of the mucosal surfaces is breached (mucosal ulcerations or periodontal disease).

Spleen Disease

Removal of the spleen predisposes patients to post-splenectomy sepsis (PSS), a condition characterized by the onset of bacteremia without a primary site of infection. Characteristically, the bacteremia is followed by the sudden onset of nausea and vomiting, meningitis, disseminated intravascular coagulation, acute respiratory distress syndrome, shock, and death in less than 72 hours. Although infants and children are primarily affected, this risk also extends to teenagers and adults. A heightened susceptibility to PSS typically exists for 1–2 years after splenectomy; however, PSS has been reported 5 and even 25 years post-splenectomy. As a consequence, the provision of antimicrobial prophylaxis is recommended for those asplenic patients who are at greatest risk for developing infection prior to undergoing a bacteremia-inducing dental procedure.

Neurologic System

Headaches

From a medical standpoint, the significance of headache is often obscure; however, it may be a symptomatic expression of tension and fatigue. The degree of incapacity, location, duration, and time-intensity curve of

headaches, in both the attack itself and its historical pattern, are most useful in establishing a diagnosis.

Dizziness and Fainting

Loss of consciousness secondary to transient ischemia to the brain and a generalized weakness of muscles characterize syncope. In contrast, the term faintness refers to a lack of strength with a sensation of impending loss of consciousness. Two-thirds of syncope episodes are autonomic reflex responses to a specific trigger such as emotional or orthostatic stress. These conditions rarely appear when the patient is recumbent. Pallor is an early and invariable finding. The onset is deliberate. Injury from falling is rare and the return to consciousness is prompt.

Seizures

A seizure may occur any time regardless of the position of the patient. The patient's color usually does not change. The attack is sudden, and if an aura is present (characterized by seeing intermittent lights or hearing a continuous buzzing sound), it rarely lasts longer than a few seconds before unconsciousness. Injury from falling is frequent because protective reflexes are abolished. Tonic-clonic convulsive movements may be a feature and result in an increased period of unconsciousness. Mental confusion, headache, and drowsiness are common.

Paresthesia and Numbness

A history of pain, skin hypersensitivity, muscle tenderness, and a combination of dysesthesia and paresthesia should alert a clinician to a possible underlying nutritional, toxic, or metabolic abnormality.

Neuralgia

The most striking form of neuralgia to affect the head and neck area involves the trigeminal nerve and is known as tic douloureux. This occurs in middle-aged and elderly individuals and causes excruciating paroxysms of pain involving the lips, gums, cheek, or chin, and rarely, the distribution of the ophthalmic division of the fifth cranial nerve. Glossopharyngeal neuralgia is a syndrome that resembles trigeminal neuralgia in many respects. The pain is intense and paroxysmal; it originates in the throat, approximately in the tonsillar fossa. In some cases the pain is localized to the ear or may radiate from the throat to the ear. Spasms may be initiated by swallowing.

Paralysis

The most common disease affecting the facial nerve is Bell's palsy. This disease is thought to result from an inflammatory reaction in or around the facial nerve near the stylomastoid foramen. The onset is acute and the paralysis may evolve over a period of several hours. Bulbar palsy results from weakness or paralysis of the muscles supplied by the medulla oblongata. A syndrome of dysphasia and dysphonia secondary to complete interruption of the intracranial portion of the vagus nerve results in a characteristic paralysis. In this situation, the soft palate droops and does not rise in phonation. There is a loss of the gag reflex on the affected side, the voice is hoarse and slightly nasal, and the vocal cord lies immobile in a cadaveric position. Paralysis resulting from a cerebrovascular accident (CVA) may involve the mouth, arms, and hands, thereby making it difficult to wear prostheses and maintain good oral hygiene. Patients with a history of CVA are likely to be taking oral anticoagulants. Since CVAs are almost always associated with evidence of cardiovascular disease, patients may be taking other medications as well.

Psychiatric Problems

Anxiety

The stress associated with contemporary society, along with the prospect of a real or

imaginary illness, is thought to induce anxiety. When this occurs in a clear relationship to a stressful event or situation, it can be accepted as normal. Only when it is excessively intense and uncontrollable or accompanied by derangement of visceral function does it become the basis for medical intervention. The anxiety state is characterized by a subjective feeling of fear and uneasy anticipation with the physiologic accompaniments of strong emotion. Behavioral clues include fidgeting and hyperactivity or rigidity, facial expressions of panic and stammering, or speech blocks. In adults, behavioral displays are frequently masked because they are perceived as socially inappropriate or embarrassing. Despite the potential limitations of patient self-report, behavioral clues remain an important component of anxiety assessment. Asking patients how they feel, in a manner that avoids the appearance of being evaluative or judgmental, often facilitates disclosure of anxiety.

Phobia

According to the DSM-III diagnostic criteria for phobic disorders, a simple phobia is a persistent, irrational fear of and compelling desire to avoid an object or a situation other than agoraphobia. Affected patients often exhibit a single phobia that involves a fear of animals, illness, heights, or closed spaces. The most common simple phobia seen in dentistry is dental phobia. It is a persistent and recurrent fear of dentistry. The patient recognizes it as unreasonable, tries to resist avoidance behavior, yet the fear is not under voluntary control. Surveys show that up to 10% of the population may have an intense fear of dentistry. Factors associated with dental phobia include unfavorable attitudes toward dentists, infrequent visits to the dentist, and dissatisfaction with appearance.

Depression

Most individuals experience periodic episodes of discouragement and despair throughout life. As with anxiety, depression is appropriate in a given life situation and is a natural, healthy reaction that is seldom the basis for medical consultation. According to the DSM-III diagnostic criteria for affective disorders, a major depressive episode is a cluster of depressive symptoms characterized by the presence of low mood and at least four of the following: (1) poor appetite or significant weight loss, (2) insomnia, (3) agitation or retardation, (4) loss of interest in usual activities, (5) loss of energy, (6) feelings of self-reproach or guilt, (7) decreased ability to concentrate, and (8) death wishes or suicidal ideation. It is the presence of these accompanying symptoms that helps to differentiate a significant depressive condition from the low mood or depressed feelings that most individuals experience from time to time, often in the context of an environmental stress.

Growths or Tumors

Radiotherapy and Chemotherapy

Proper oral health is important for patients undergoing cancer therapy. When oral health is neglected, the oral complications resulting from cancer therapy are more severe. Good oral hygiene and elimination of existing or potential sources of infection or irritation will minimize mucositis, infection, hemorrhage, xerostomia, neurologic disorders, and nutritional problems and improve the patient's quality of life.

Conclusion

In eliciting and interpreting the historical profile, the clinician must exercise a degree of skill, care, and judgment ordinarily possessed by other members of the profession in similar circumstances. A breach of this duty constitutes negligence, for which the dentist may be held liable. Although consultation with a patient's physician(s) is desirable in

many cases, it is the dentist, not the physician, who is responsible for the physical and emotional well-being of the patient undergoing dental treatment.

In addition to gathering, documenting, and interpreting data about the patient's health, the dentist also is charged with maintaining confidentiality. The need to maintain confidentiality extends to the office staff as well. A breach of that duty opens the dentist to the possibility of a damage claim in tort law. The patient must give consent before any information can be disclosed to a third party unless allowed or required by law.

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3 Basic Procedures in Physical Examination

Time-tested basic procedures in physical examination include inspection, palpation, percussion, auscultation, olfaction, and evaluation of function. The process begins the moment the patient presents for care. Note general appearance, body language, and mannerisms, as these are reliable indicators of the patient's physical and mental state. When shaking the patient's hand, does the patient respond in kind? A patient who shuns a handshake may be doing so for cultural reasons, or the handshake may be painful, as for a patient suffering from arthritis. Does the patient maintain or avoid eye contact when spoken to? Reticence to maintain eye contact and poor personal hygiene may indicate depression or some other psychological disorder.

Establish the patient's level of consciousness, cognitive function, and language comprehension within the first minute or 2 of contact with the patient. When the patient speaks, note voice quality. Abnormalities in volume may indicate hearing loss, while hoarseness may be due to laryngeal pathoses. Peculiarities of speech such as an unusual accent or pattern of communication, slurring, dysphasia, aphasia, garbled speech, or lapses of speech may be consequential observations.

Inspection

Inspection is defined as the process of examination that relies on the sense of vision. It is not only the most common but often the most successful examination technique.

Note the patient's physical characteristics, that is, anatomical architecture, mobility, gait, color, and respiratory function. Alterations in body size, shape, and symmetry may suggest developmental or acquired abnormalities. Mobility, gait, and postural abnormalities may indicate skeletal or neuromuscular defects. Pallor may be an indicator of anemia, while cyanosis may indicate problems associated with the respiratory and/or cardiovascular system. Jaundice may be a sign of hemolytic anemia, liver disease, or a pancreatic abnormality. Evidence of respiratory difficulty may be associated with allergic, pulmonary, or cardiac disorders.

Once global inspection of the patient is accomplished, the clinician should proceed to the more focused inspection of specific tissues such as visible skin and the oral mucosa. The skin (Figure 3.1) and oral mucosa (Figure 3.2) are metabolically active tissues. Both serve a primary protective function for the body, and through their rich neural and vascular supply mediate sensory contact with the environment and help regulate temperature.

To the uninitiated, many skin and mucosal lesions may look alike, but most have characteristic primary presentations. These primary lesions often progress to form well-defined secondary lesions as a consequence of such factors as normal maturation, trauma, therapy, or secondary infection. For example, a lip lesion may begin as a vesicle, but may quickly break down to form an erosion or ulcer and eventually crust as healing occurs. There is often a dynamic spectrum of coexisting primary and secondary lesions.

Careful inspection of the skin and oral mucosa may reveal the first signs of internal or systemic disease and, on occasion, skin lesions may provide the first clues essential for the diagnosis of certain oral conditions. Note changes in color (pigmentation, vascularity); the presence of edema, swelling, and bulging; surface characteristics such as moistness, dryness, or oiliness; and other unusual findings. The terminology used to describe mucocutaneous lesions is not only descriptive but may at times be suggestive of the underlying cause. The pattern of distribution of the various lesions is also important and may be described as linear, annular (in a ring), or serpiginous (in a curvilinear pattern, serpent-like).

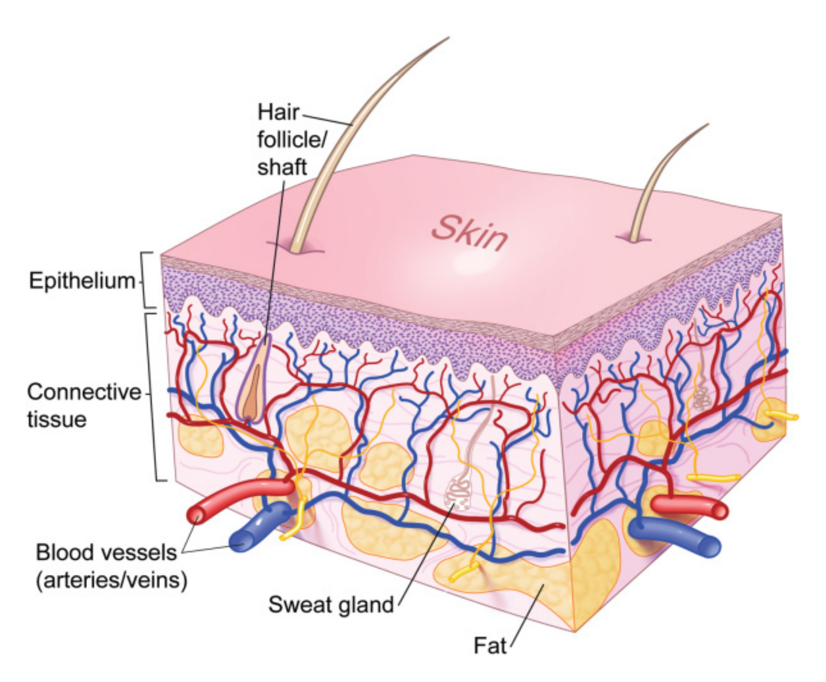


Figure 3.1. Normal skin.

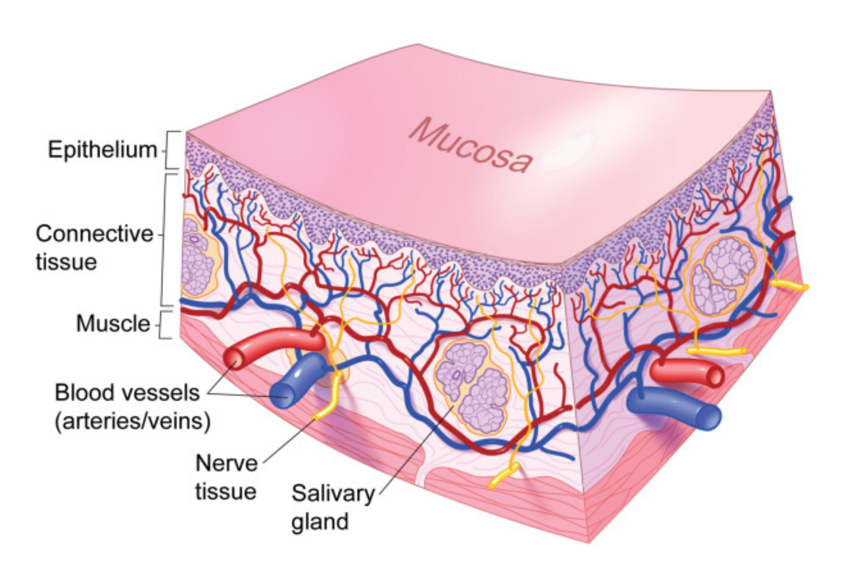


Figure 3.2. Normal mucosa.

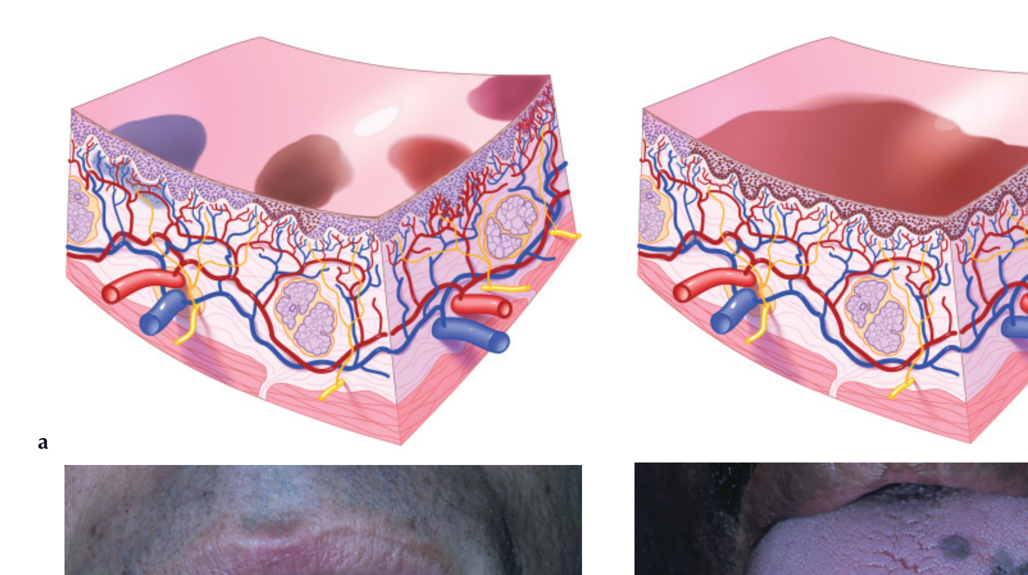
Primary Lesions of the Skin and Oral Mucosa

Macule

A macule (Figures 3.3a and 3.3b) is a circumscribed, flat lesion less than 1 cm in size, varied in shape and color, and may represent hyperpigmented, hypopigmented, or vascular abnormalities. Figure 3.3b is an example of multiple macules on the lower lip due to physiologic pigmentation.

Patch

A patch (Figures 3.4a and 3.4b) is a circumscribed, flat lesion larger than 1 cm in size, varied in shape and color, and may represent hyperpigmented, hypopigmented, or vascular abnormalities. Figure 3.4b is an example of a patch on the dorsum of the tongue due to physiologic pigmentation.



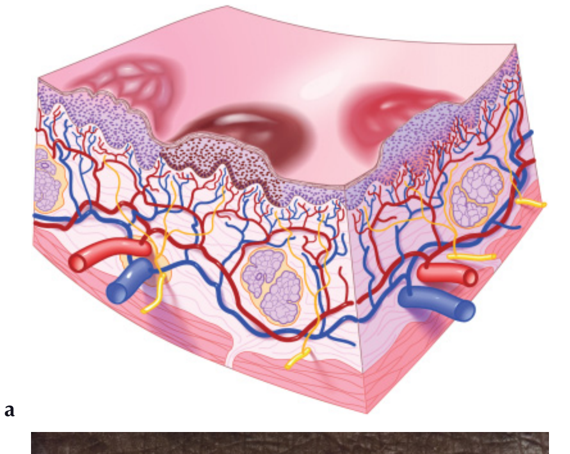
Figures 3.3a and b. Macule.



Figures 3.4a and b. Patch.

Papule

A papule (Figures 3.5a and 3.5b) is a circumscribed, elevated, superficial, solid lesion less than 1 cm in size, varied in shape and color, and may reflect hyperplasia of cellular structures or represent cellular infiltrates. Figure 3.5b is an example of a dermal papule in a patient with lichen planus.

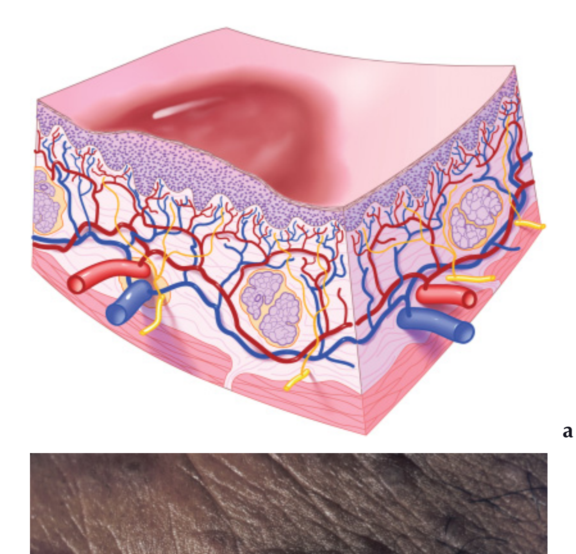




Figures 3.5a and b. Papule.

Plaque

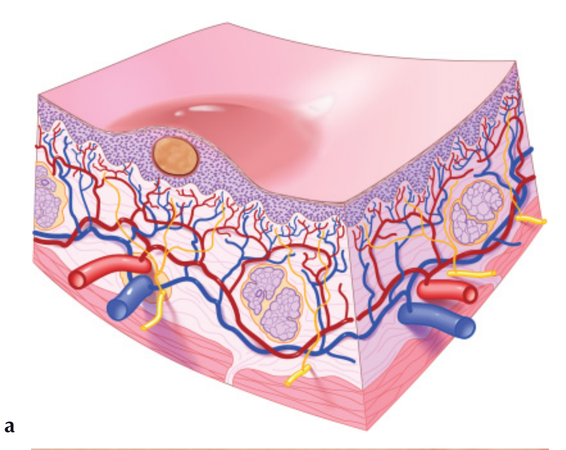
A plaque (Figures 3.6a and 3.6b) is a circumscribed, elevated, superficial, solid lesion larger than 1 cm in size, varied in shape and color, and may reflect hyperplasia of cellular structures, represent cellular infiltrates, or may be formed by a confluence of papules. Figure 3.6b is an example of a dermal plaque in a patient with lichen planus.



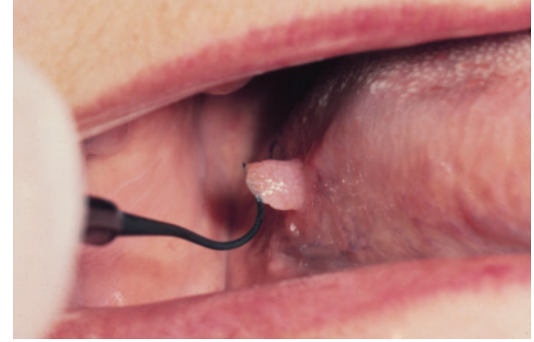
Figures 3.6a and b. Plaque.

Nodule

A nodule (Figures 3.7a and 3.7b) is a solid palpable lesion less than 1 cm in size. Its depth may be above, level with, or beneath the skin or mucosal surface. Nodules may be the result of inflammatory, neoplastic, or metabolic processes. Descriptors such as soft, firm, hard (bony), fixed, movable, pedunculated (has a stem-like connecting part, a stalk by which a nodule or a tumor is attached to normal tissue), or sessile (attached by a base; not pedunculated or stalked) are helpful when describing these lesions. Figure 3.7b is an example of a pedunculated nodule on the right lateral surface of the tongue.



a

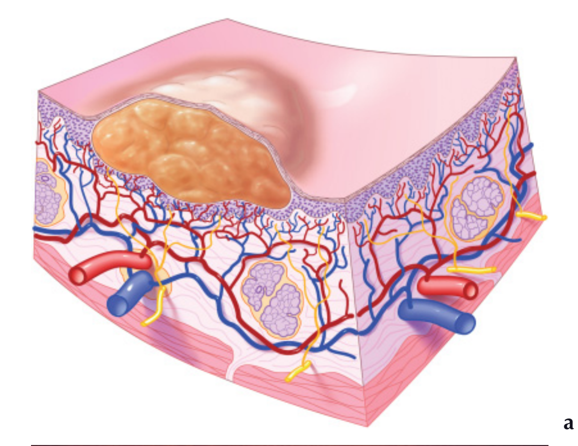


b

Figures 3.7a and b. Nodule.

Tumor

A tumor (Figures 3.8a and 3.8b) is a solid palpable lesion larger than 1 cm in size. Its depth may be above, level with, or beneath the skin or mucosa. Tumors may be the result of inflammatory, metabolic, and neoplastic processes. Descriptors such as soft, firm, hard (bony), fixed, movable, pedunculated, or



a



b

Figures 3.8a and b. Tumor.

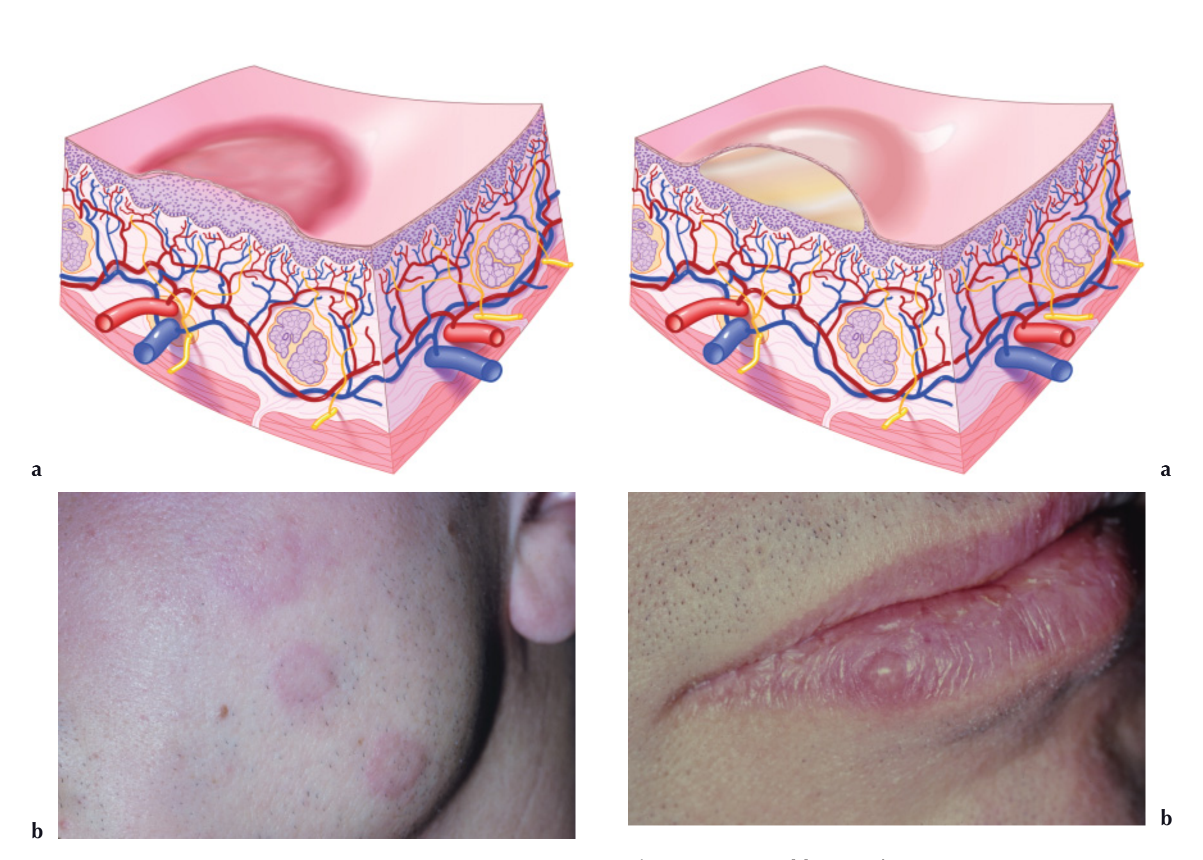
sessile are helpful when describing these lesions. Figure 3.8b is an example of a tumor affecting the dorsum of the tongue.

Wheal

A wheal (Figures 3.9a and 3.9b) is an edematous, rounded or oval transitory papule of variable size, usually the result of an allergic reaction. Figure 3.9b is an example of wheals on the face of a patient with an allergy to latex.

Vesicle

A vesicle (Figures 3.10a and 3.10b) is a circumscribed elevated intraepithelial or subepithelial lesion less than 1 cm in size, which contains a serous fluid. Figure 3.10b is an example of a vesicle associated with recurrent herpes labialis.

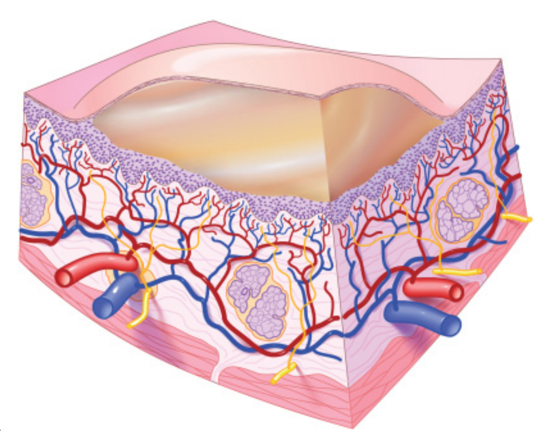


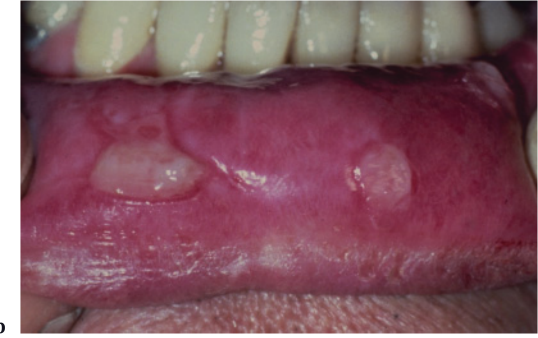
Figures 3.9a and b. Wheal.

Figures 3.10a and b. Vesicle.

Bulla

A bulla (Figures 3.11a and 3.11b) is a circumscribed elevated intraepithelial or subepithelial lesion larger than 1 cm in size, which contains a serous fluid. Figure 3.11b is an example of multiple bullae occurring on the inner aspect of the lower lip in a patient with pemphigus vulgaris.

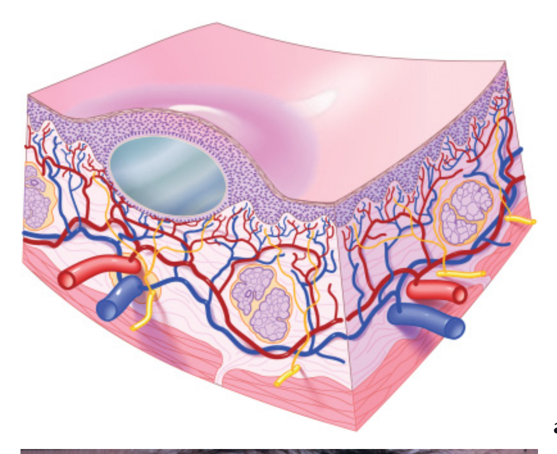

a


b

Figures 3.11a and b. Bulla.

Cyst

A cyst (Figures 3.12a and 3.12b) is an encapsulated lesion of variable size in subcutaneous or submucosal tissue filled with liquid or semisolid material. Figure 3.12b is an example of a dermoid cyst; the lesion is located about 2 cm lateral to the comissure of the mouth.

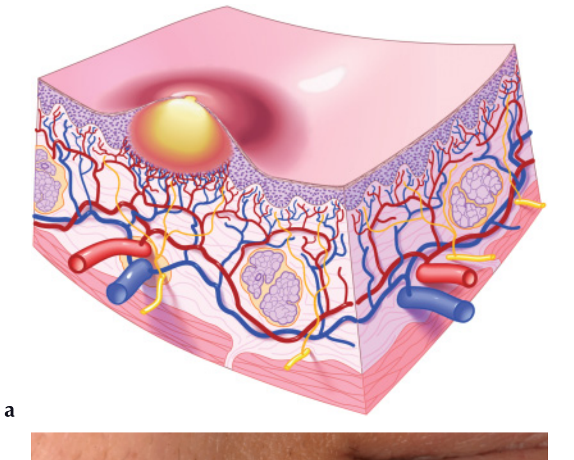

a


b

Figures 3.12a and b. Cyst.

Pustule

A pustule (Figures 3.13a and 3.13b) is a circumscribed elevation of variable size and shape containing a purulent exudate. Depending on the color of the purulent exudates, it may appear white, yellow, or greenish-yellow. Figure 3.13b is an example of a dermal pustule.



a

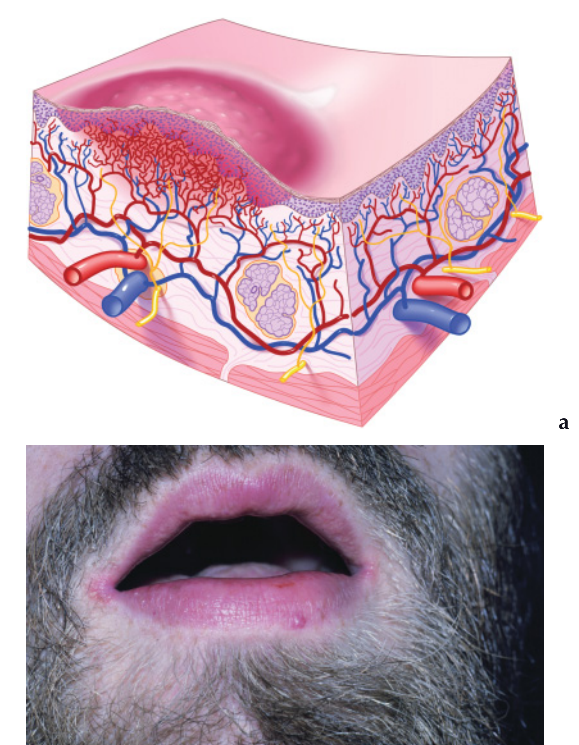


b

Figures 3.13a and b. Pustule.

Hemangioma

A hemangioma (Figures 3.14a and 3.14b) is a red irregular macule or patch of variable size and shape caused by dilation of dermal or mucosal capillaries. Figure 3.14b is an example of capillary hemangioma located on the patient's lower lip.



a

b

Figures 3.14a and b. Hemangioma.

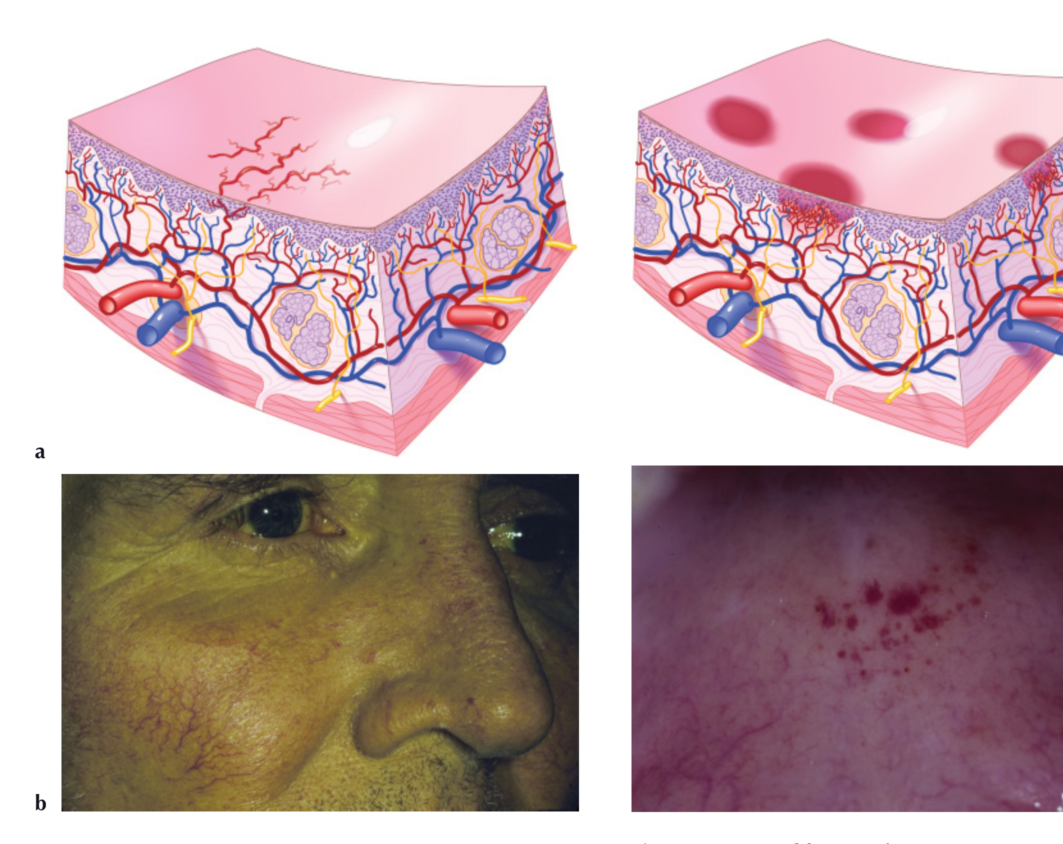
Telangiectasia

Telangiectases (Figures 3.15a and 3.15b) are serpiginous lesions caused by permanent dilation of superficial capillaries. Figure 3.15b is an example of multiple telangiectases on the face of a patient with alcoholic cirrhosis of the liver.

Secondary Lesions of the Skin and Oral Mucosa

Petechia

A petechia (Figures 3.16a and 3.16b) is a circumscribed deposit of extravasated blood or blood pigments less than 2 mm in size. Figure 3.16b is an example of multiple petechiae occurring on the soft palate in a patient taking clopidogrel, an antithrombotic agent.

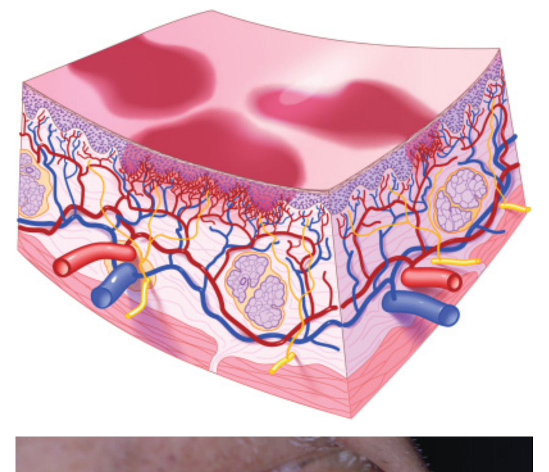


Figures 3.15a and b. Telangiectasia.

Figures 3.16a and b. Petechiea.

Purpura

A purpura (Figures 3.17a and 3.17b) is a circumscribed deposit of extravasated blood or blood pigments between 2 and 10 mm in size. Figure 3.17b is an example of a purpura on the left buccal mucosa secondary to trauma in a patient taking warfarin, an oral anticoagulant.



a

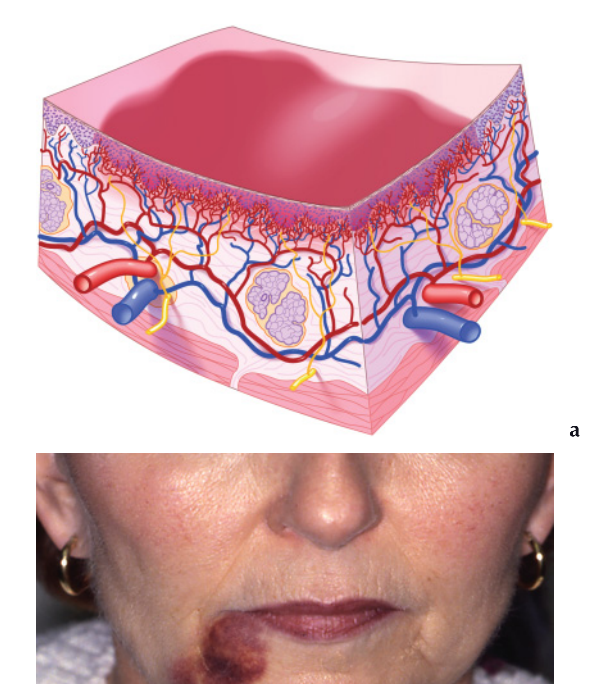


b

Figures 3.17a and b. Purpura.

Ecchymosis

An ecchymosis (Figures 3.18a and 3.18b) is a circumscribed deposit of extravasated blood or blood pigments larger than 1 cm in size. Figure 3.18b is an example of a large ecchymotic lesion secondary to trauma associated with a minor salivary gland biopsy.



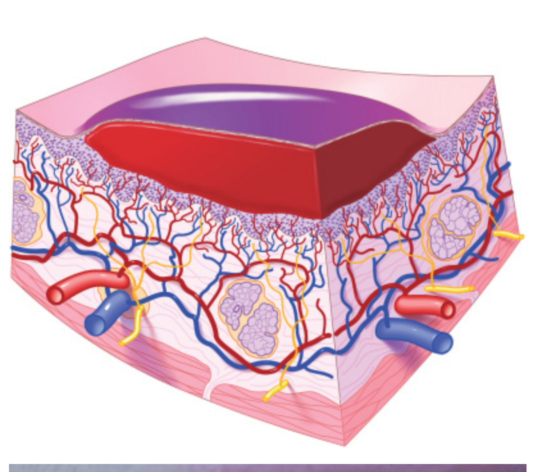
a

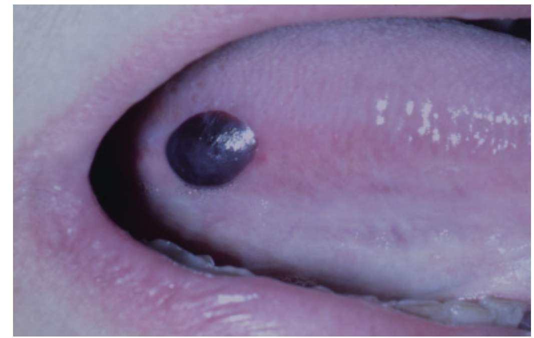
b

Figures 3.18a and b. Ecchymosis.

Hematoma

A hematoma (Figures 3.19a and 3.19b) is an accumulated mass of extravasated blood that usually clots to form a solid swelling of variable size and shape within a tissue. Figure 3.19b is an example of a hematoma affecting the lateral border of the tongue in a patient who was taking heparin, a parenteral anticoagulant.

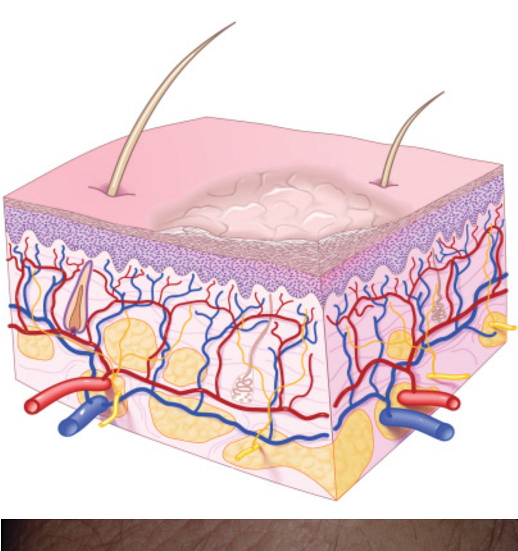

a


b

Figures 3.19a and b. Hematoma.

Scale

Scales (Figures 3.20a and 3.20b) are characterized by abnormal shedding (desquamating), usually of dry and flaky, dead epithelial cells. Figure 3.20b is an example of scaly lesions occurring on the forearm of a patient with psoriasis.


a

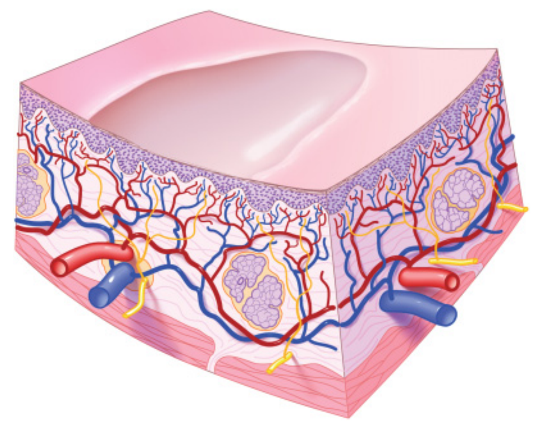

b

Figures 3.20a and b. Scale.

Atrophy

Atrophy (Figures 3.21a and 3.21b) is characterized by diminution in the size of cells, tissues, organs, or body parts. Figure 3.21b is an example of atrophy of the filiform papillae occurring on the dorsum of the tongue.

a



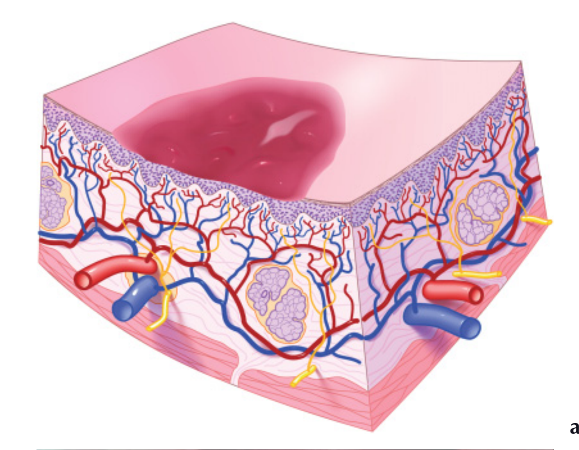
b



Figures 3.21a and b. Atrophy.

Erosion

Erosion (Figures 3.22a and 3.22b), as it relates to skin and oral mucosa, is characterized by a gradual breakdown (denudation) of the epithelium, which heals without scarring. Erosion, as it relates to oral hard tissues (enamel, dentin, cementum), is characterized by gradual loss of tooth substance by a chemical process that does not involve a known bacterial action. Figure 3.22b is an example of erosions affecting the marginal gingiva of the maxillary right premolar in a patient with erosive lichen planus.



a

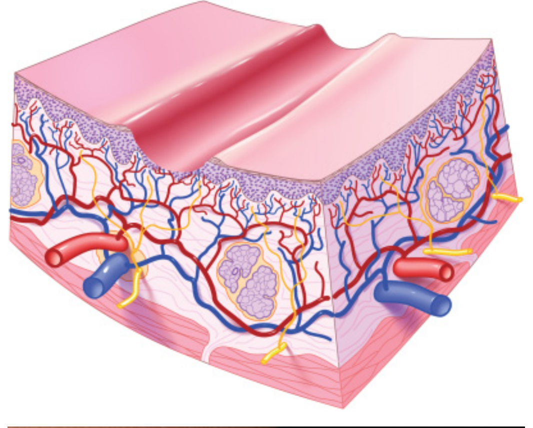


b

Figures 3.22a and b. Erosion.

Excoriation

Excoriation (Figures 3.23a and 3.23b) is a superficial, sometimes linear excavation of the epidermis usually associated with scratching. Figure 3.23b is an example of excoriation of the epidermis of the forearm.



a

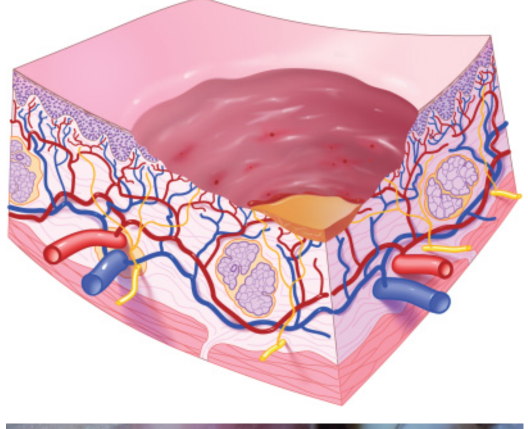


b

Figures 3.23a and b. Excoriation.

Ulcer

An ulcer (Figures 3.24a and 3.24b) is an irregularly shaped excavation of the epithelium that extends below the basal cell layer and may heal with a scar. Figure 3.24b is an example of a recurrent aphthous ulceration occurring on the labial mucosa.



a



b

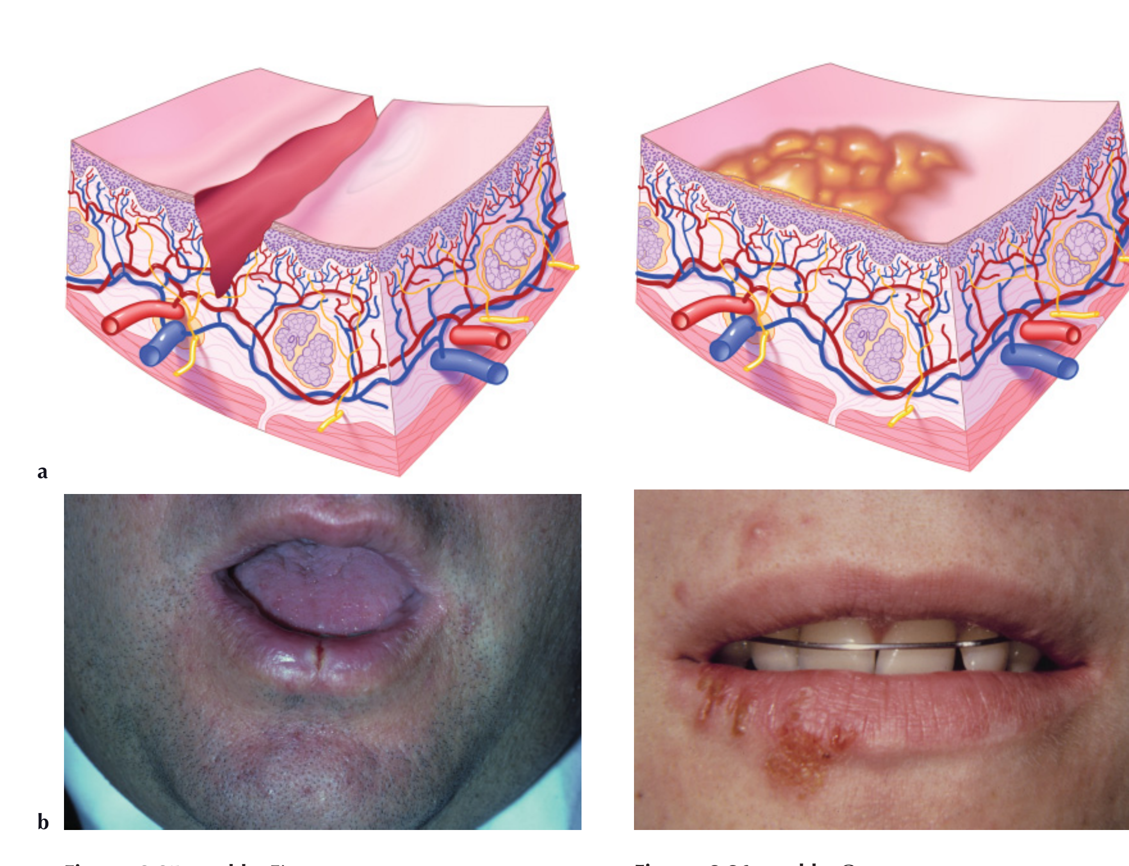
Figures 3.24a and b. Ulcer.

Fissure

A fissure (Figures 3.25a and 3.25b) is a linear crack or cleavage in epithelial tissue with sharply defined abrupt walls. Figure 3.25b is an example of a self-induced fissure occurring at the midline of the lower lip.

Crust

A crust (Figures 3.26a and 3.26b) is a hardened deposit of variable thickness consisting of dried blood, serum, or purulent exudate on skin and lip vermilion. Figure 3.26b is an example of crusting that has occurred after the rupturing of vesicles in a patient with recurrent herpes labialis.

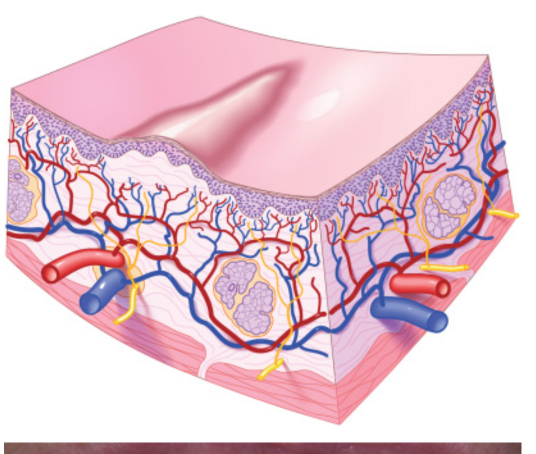


Figures 3.25a and b. Fissure.

Figures 3.26a and b. Crust.

Scar

A scar (Figures 3.27a and 3.27b) is a mark that remains after the healing of a wound, which may be atrophic or hypertrophic as a consequence of variable degrees of collagen proliferation. Figure 3.27b is an example of a scar affecting the middle of the lower lip after injury sustained from a fall.



a

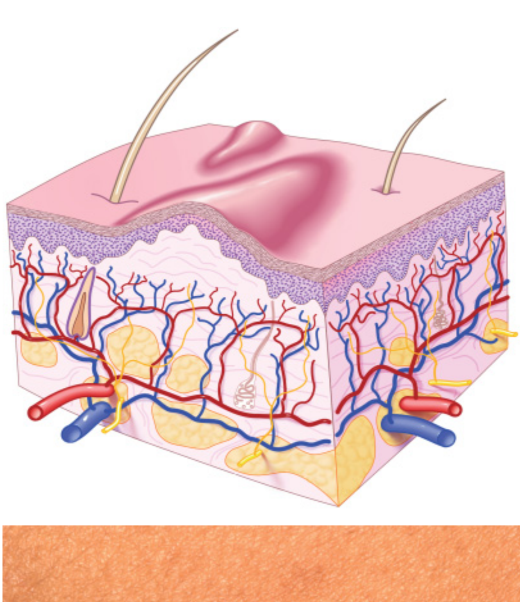


b

Figures 3.27a and b. Scar.

Keloid

A keloid (Figures 3.28a and 3.28b) is a sharply elevated, irregularly shaped hypertrophic scar due to the formation of an excessive amount of collagen, which tends to extend and grow beyond the original site of injury during connective tissue repair. Figure 3.28b is an example of keloid occurring on the skin.



a

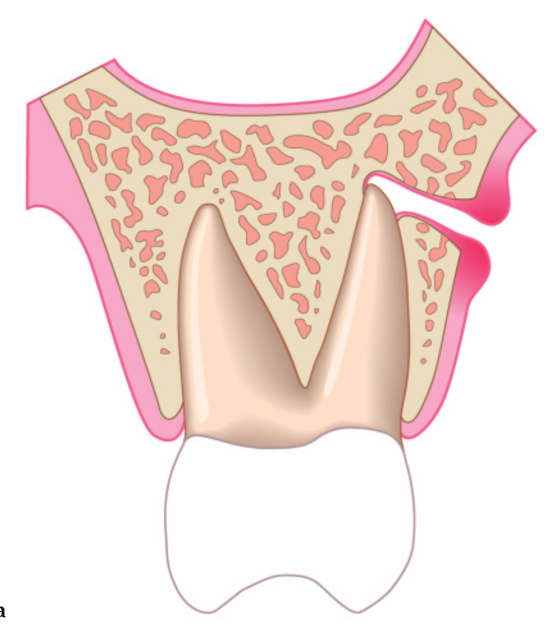


b

Figures 3.28a and b. Keloid.

Sinus

A sinus (Figures 3.29a and 3.29b) is an abnormal channel that leads from a pathological space to an anatomical space permitting the escape of pus. Figure 3.29b is an example of a sinus tract associated with an abscessed maxillary tooth.


a


b

Figures 3.29a and b. Sinus.

Special Lesions

Comedone

Comedones are plugs of whitish or blackish sebaceous and keratinous material lodged in pilosebaceous follicles commonly seen on the face.

Clubbing of the Nails/Fingers

Clubbing (Figures 3.30a and 3.30b) is characterized by curved overgrowth of the nail bed with bulbous enlarged fingertips. It is usually associated with congenital heart defects, congestive heart failure, chronic obstructive pulmonary disease, and carcinoma of the lung. Figures 3.30a and 3.30b represent clubbing of the fingernails in a patient who was diagnosed with cardiovascular disease.


a


b

Figures 3.30a and b. Digital clubbing.



Figure 3.31. Onycholysis.

Onycholysis

Onycholysis (Figure 3.31) is the result of keratin deposition beneath the nail bed, which produces opacification and irregular separation of the distal portion of the nail. Figure 3.31b is an example of onycholysis of the fingernails in a patient who had psoriasis.

Palpation

Palpation is defined as the process of examination that relies on the sense of touch. The classic application of this examination technique is reflected in the process of assessing a patient's pulse pressure (reflecting the numerical difference between systolic and diastolic BP), rate, and rhythm (see "Evaluation of Function").

In the head and neck area, palpation is invaluable in confirming and expanding upon the observations noted on inspection. It should be initiated as a light touch, followed by deep palpation. A light touch is useful to detect surface characteristics such as tissue texture and temperature, as well as qualities such as pulsation or fluctuation; deep palpation confirms the extent of nodules or tumors (Table 3.1).

Table 3.1. Tissue characteristics noted upon palpation.

Temperature—heat
Tenderness or pain—superficial, deep, rebound, referred
Muscle tones—increased resistance, spasm, rigidity
Mass—nodule, tumor, and lymph node
Location—relation to other tissues
Architecture—size, shape, symmetry, discreteness
Consistency—soft, firm, hard (bony)
Mobility—attachment (bound or unbound)
Quality—pulsating or fluctuating

The palpation of certain anatomical structures requires a bidigital or bimanual technique—for example, when palpating for cervical lymph nodes, the area being assessed is gently pinched between two fingers (bidigital palpation); when palpating the floor of the mouth, the structures being examined are trapped between the fingers of each hand (bimanual palpation). As palpation may cause the patient to experience tenderness or pain, this possibility mandates judicious application of the technique.

Percussion

Percussion is defined as the process of examination that relies on the technique of gently tapping an area of the body while noting the resonance or sound produced and the resistance encountered. It is the primary physical maneuver used in medicine to detect the presence or level of pleural effusion.

In the head and neck area, percussion is often used to provoke pain in an effort to identify teeth with periodontal or pulpal disease in patients who are unable to localize the discomfort or pain to a specific tooth. Similarly, percussion is useful to evaluate tenderness of the maxillary sinuses (maxillary sinusitis), which may be referred maxillary teeth.

Auscultation

Auscultation is defined as the process of examination that usually involves listening to the surface of the body with the aid of a stethoscope.

Auscultation is the time-tested technique used when determining blood pressure (see "Evaluation of Function") and breathing sounds such as inspiration, expiration, rales, rhonchi, stridor, and wheezing (see "Evaluation of Function"). When assessing sounds, such characteristics as intensity, pitch, duration, and quality should be also determined. In order to assess sounds adequately, these observations should be made in a quiet environment. Auscultation of the temporomandibular joint may reveal crepitus, the crackling or grating sound produced by bone rubbing on bone or roughened cartilage.

Olfaction

Olfaction is defined as the process of examination that relies on the sense of smell. It is useful to detect odors arising from the patient that may suggest the presence of local or systemic disease (Table 3.2).

Evaluation of Function

Pulse Rate and Rhythm

The pulse is a series of pressure waves within an artery caused by contractions of the left ventricle and corresponds to the heart rate.

The pulse rate and rhythm vary with the demand for oxygen, age, and various disease states (Table 3.3). Consequently, it should be recorded for all new patients at the time of initial appointment and it should be recorded at all subsequent appointments for all patients with a history of cardiac arrhythmias, hypertension, cardiovascular diseases, diabetes mellitus, thyroid disorders, adrenal disease, renal dysfunction, and significant use of tobacco and coffee.

The normal pulse rate is 60–100 beats per minute for adults, 90–120 beats per minute for children, and 70–80 beats per minute in the aged.

Technique

To determine the pulse rate and rhythm, the patient's hand is grasped with the palm facing upward. The three middle fingers of the examiner are placed on the radial artery (located at the patient's wrist, lateral to the radius) with the index finger nearest to the heart (Figure 3.32). With the fingers in this

Table 3.2. Common conditions associated with halitosis.

Oral conditions	Oral sepsis associated with extensive caries, stomatitis, gingivitis, or periodontal disease Fusospirochetal infections associated with ulcerative gingivitis, pharyngitis, or cancrum oris
Systemic conditions	Upper respiratory tract infections Lung abscess or bronchiectasis suggested by a fetid, foul putrefactive breath Diet, gastrointestinal disturbances Diabetic acidosis or hyperglycemic coma associated with a sweet, fruity acetone odor Renal failure (uremia) associated with an odor of ammonia Liver failure characterized by fetor hepaticus, a mousy, musty odor Anxiety indicated by odor of alcohol resulting from self-medication on a sedative or compulsive basis

Table 3.3. Alterations in pulse rate and rhythm and associated conditions.

Sinus bradycardia Impulses originate from the SA node at a slow rate as a result of increased vagal tone.	HR is <60 beats per minute and the rhythm is regular. May be asymptomatic or cause light-headedness, fainting, chest discomfort, hypotension, and dyspnea.	Common in athletes and in patients with hypothyroidism and increased intracranial pressure, and during treatment with drugs with negative chronotropic action (e.g., β_1-adrenergic receptor antagonists, calcium channel blocking agents and digoxin).
Sinus tachycardia Impulses originate from the SA node at a rapid rate under the influence of increased sympathetic tone or vagal blockade.	HR is 100–180 beats per minute and the rhythm is regular. May be asymptomatic or cause palpitations (sensa-tion of skipped beats or rapid forceful beats) or symptoms of hemodynamic compromise (dyspnea, chest discomfort, syncope).	Common in patients after exercise or smoking; in patients with hyperthyroidism, anxiety, toxic states, fever, anemia, severe hemorrhage, debilitation, and acute or chronic heart disease; and in patients taking stimulants (e.g., tea, coffee) and medications with positive chronotropic effects.
Atrial flutter Impulses originate from a single abnormal focus in the atria resulting in the circular propagation of the impulses in the atria.	Heart rate 250–350 beats per minute, and the rhythm is regular. Because the AV node is unable to transmit all of the impulses, only about half will get through, resulting in a ventricular rate of 150 beats/min. May be asymptomatic or cause palpitations, chest discomfort, dyspnea, weakness, and syncope.	Much less common that atrial fibrillation, but its causes and hemodynamic consequences are similar (see below).
Atrial fibrillation Impulses originate from multiple atrial foci, which travel in a random manner in the atria.	Heart rate 350–450 beats per minute, and the rhythm is irregular. The ventricles respond to only about 120–180 of the impulses. Symptoms include palpitations, vague chest discomfort, weakness, light-headedness, dyspnea. Stasis of blood in the fibrillating atrium can lead to blood clot formation and systemic embolism, which may present as stroke-like illness, i.e., sudden confusion; acute painful, pale, pulseless limbs; and an acute abdomen.	Common causes include hypertension, cardiomyopathy, mitral or tricuspid valvular disorders, hyperthyroidism, and binge alcohol drinking. Less common causes include pulmonary embolism, congenital heart defects, COPD, myocarditis, and pericarditis.
Premature ventricular contractions (PVCs) Impulses originate from an ectopic ventricular focus.	Pronounced pause in an otherwise normal rhythm. May occur erratically or at predictable intervals, e.g., every second (bigeminy) or third (tigeminy) beat.	PVCs may be an occasional finding in otherwise healthy adults and the incidence increases with age, fatigue, emotional stress, and the use of coffee and tobacco. PVCs are significant in a patient with cardiovascular disease (coronary heart disease, valvular disease, hypertension, and congestive heart failure).

Table 3.3. *Continued*

Ventricular tachycardia (VT) Usually evolves from an ectopic focus in the right or left ventricle.	Rate 120–220 beats per minute. Sustained VT is almost always symptomatic, causing palpitations, fatigue, light-headedness, syncope, or sudden cardiac death.	It is a serious arrhythmia that occurs in patients with organic heart disease (prior MI and cardiomyopathy). May be precipitated by drugs such as digoxin, serotonin reuptake inhibitors, and tricyclic antidepressants.
Ventricular fibrillation (VF) The myocardium depolarizes in a chaotic manner and coordinated ventricular activity ceases.	Heart rate 350–450 beats per minute and the rhythm is irregular. The heart ceases to pump, the blood pressure falls, and unconsciousness occurs. If left untreated, death will follow in about 3–5 minutes.	Coronary artery disease is the most common cause of VF, followed by hypertrophic or dilated cardiomyopathy. VF is the presenting rhythm for about 70% of patients in cardiac arrest.
Atrioventricular (AV) blocks Partial or complete interruption of impulse conduction from the atria to the ventricles.	First degree Delay in impulse conduction Asymptomatic Second degree Intermittent failure in conduction Asymptomatic or experience light-headed-ness and syncope Third degree Permanent failure in conduction. Cardiac function is maintained by a ventricular pacemaker Symptoms may include light-headedness, fatigue, syncope, and heart failure	Common causes are idiopathic fibrosis and sclerosis of the conduction system (50%) and ischemic heart disease (40%). The rest are due to drugs (e.g., β-blockers, digoxin), increased vagal tone, valvulopathy, and congenital heart disorders.

position, gentle pressure is applied to feel the pulse rate and rhythm for a full minute.

Figure 3.32. Pulse pressure.

Blood Pressure

Blood pressure (BP), defined as the lateral pressure exerted by the blood in a unit area of blood vessel wall, is a function of cardiac output and peripheral vascular resistance.

The BP is a reliable indicator of cardiovascular function and correlates well with a number of other systemic diseases and conditions (Table 3.4). Consequently, the BP should be recorded on all new patients at the time of initial appointment and at all subsequent appointments on all patients with a history of hypertension, cardiovascular diseases, diabetes mellitus, thyroid disorders,

Table 3.4. Common conditions that affect BP.

Etiology of hypotension	Etiology of hypertension
Anemia	Physical or emotional stress
Hypoaldosteronism	Hyperaldosteronism
Hypothyroidism	Hyperthyroidism
Syncope	Dyslipidemia
Shock	Diabetes mellitus
Heart failure	Renal dysfunction
Collagenvascular disorders	Steroid therapy

Table 3.5. Cuff selection.

Patient size	Arm circumference	Cuff bladder size
Child or small adult	< 23 cm	12 cm × 18 cm
Standard adult	< 33 cm	12 cm × 26 cm
Large adult	< 50 cm	12 cm × 40 cm

adrenal disease, renal dysfunction, and significant use of tobacco, coffee, and alcohol.

BP is classified as normal (< 120/80 mm Hg), prehypertension (120–139/80–89 mm Hg), stage 1 hypertension (140–159 mm Hg systolic or 90–99 mm Hg diastolic), or stage 2 hypertension (≥ 160 mm Hg systolic or ≥ 100 mm Hg diastolic).

Technique

In the everyday practice of medicine (including medical dentistry), the BP is determined by sphygmomanometry, using a combination of the palpatory and auscultatory methods. A sphygmomanometer consists of a pressure manometer (mercury-gravity or aneroid type), a compressor cuff, and a pressure source. Automated readers that are available should be avoided, as they are often inaccurate.

The mercury-gravity manometer consists of a uniform diameter straight glass tube with a reservoir containing mercury. The pressure chamber of the reservoir communicates with the compression cuff through a rubber tube. When pressure is exerted on the mercury in the reservoir it falls, and the mercury in the glass tube rises. Since the weight of the mercury is dependent on gravity, which is constant, a given amount of pressure will always support a column of mercury of the same height. The mercury-gravity manometer is the most accurate, does not require recalibration, and is the standard for measuring BP.

The aneroid manometer consists of a metal bellows, which is connected to the compression cuff. Variations of pressure within the system cause the bellows to expand and collapse. The movement of the bellows rotates a gear that turns a needle, pivoted on bearings, across a calibrated dial. Since the blood pressure recorded with the aneroid manometer depends upon the elasticity of the metal bellows, it is subject to errors inherent in the elastic properties of metals. For this reason, the aneroid manometer must be calibrated against a mercury manometer at regular intervals.

The compressor cuff consists of an inflatable rubber bladder enclosed in an inelastic covering and the pressure source consisting of a rubber hand bulb and pressure control valve. An appropriately sized cuff should cover two-thirds of the biceps; its bladder should be long enough to encircle more than 80% of the arm and should have a width that equals at least 40% of the arm's circumference. Thus, children require smaller cuffs and obese patients require larger cuffs (Table 3.5).

Ideally, the BP is measured after the patient has rested comfortably for at least 5 minutes in a sitting or recumbent position. The examiner's chair should be arranged so that the patient's right arm is always and inevitably presented for recording the BP. The arm should be abducted, slightly flexed, and supported by a smooth, firm surface. If the arm is unsupported, the BP may be elevated by 10–12 mm Hg due to added hydrostatic pressure induced by gravity. The brachial artery over which the blood pressure is to be

recorded should be at a level with the heart (Figure 3.33).

The deflated compression cuff is applied snugly around the right arm. The lower edge of the cuff should be 2–3 cm above the antecubital fossa. The radial pulse is palpated and the rate is noted. The compression cuff is then inflated (when the cuff is inflated it should not bulge nor become displaced) to about 30 mm Hg above the pressure at which the radial pulse disappears. The cuff is then deflated at a rate of 2–3 mm Hg per heartbeat. The level of pressure at which the pulse in the radial artery returns is noted and recorded as the systolic BP. The diastolic BP is difficult to measure by palpation and is not generally determined by this method.

After the systolic pressure has been determined by the palpatory method, the BP is then determined by auscultation over the brachial artery at a point 2–3 cm below the edge of the compression cuff (in the antecubital fossa). The brachial artery is first palpated, and then the bell of the stethoscope is applied lightly but snugly over it to produce an airtight seal. The bell must not come in contact with the patient's clothing or with the compression cuff.

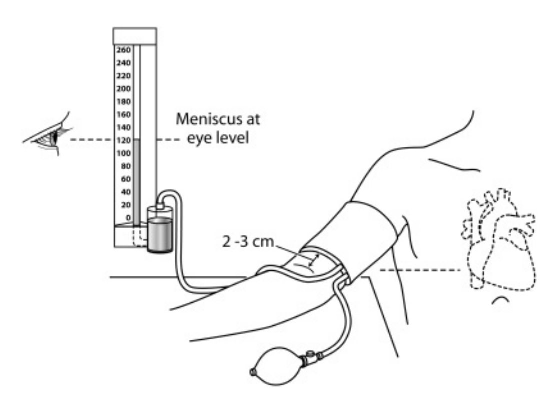


Figure 3.33. Blood pressure.

The compression cuff is inflated rapidly to about 30 mm Hg above the systolic pressure as previously determined by the palpatory method. The cuff is then deflated at a rate of 2–3 mm Hg per heartbeat. While watching the meniscus of the mercury column of the mercury-gravity manometer or the needle of the aneroid manometer, the pressure at which characteristic changes in the Korotkoff sounds (Figure 3.34) occur is noted. From the changes in the quality of the sounds, the systolic and diastolic BP is determined.

Systolic BP

The pressure within the compression cuff as indicated by the level of the mercury column (mercury-gravity manometer) or the position of the needle (aneroid manometer) at the moment the Korotkoff sounds are first heard represents the systolic BP. This is the start of Phase 1, which begins with faint, clear, and rhythmic tapping or thumping sounds that gradually increase in intensity.

Diastolic BP

The pressure within the compression cuff indicated by the level of the mercury column (mercury-gravity manometer) or the position

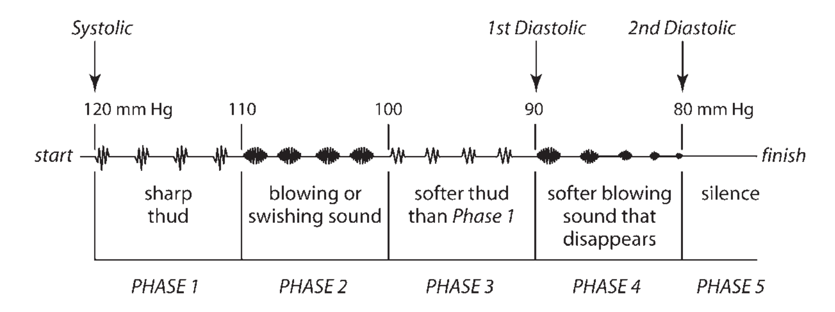


Figure 3.34. Korotkoff sounds.

Table 3.6. Abnormal rate and depth of respiration and associated conditions.

Tachypnea	Increased rate and decreased depth of respiration.
Hyperpnea	Increased rate and depth of respiration typical of hyperventilation syndrome.
Kussmaul-Kien respiration	Hyperpnea—30–40 breaths per minute typical of profound diabetic acidosis and hyperglycemic coma.
Cheney-Stokes breathing	Hyperpnea alternating with periods of apnea typical of profound toxicity associated with heart disease, chronic nephritis, and advanced brain tumor.
Prolonged inspiration (sigh)	Typical of anxiety or a precursor to hyperventilation.
Prolonged expiration	Typical of moderate-to-advanced pulmonary emphysema.

of the needle (aneroid manometer) at the moment the Korotkoff sounds become muffled represents the first diastolic pressure (beginning of Phase 4). The second diastolic pressure is the pressure within the compression cuff at the moment the sounds finally disappear (beginning of Phase 5). Disappearance of the sound marks the diastolic BP.

Pulse Pressure

The pulse pressure reflects the numerical difference between the systolic and the diastolic BP. The "hammering" or "pounding" effect of elevated pulse pressure (noted when palpating the radial artery) damages arterial walls, contributes to arteriosclerosis, and leads to target organ damage. The pulse pressure closely correlates with the systolic BP and is a reliable cofactor that will provide further evidence to either confirm or rule out significant cardiovascular disease.

Respiration

Respiration is the process of gaseous exchange between an organism and its environment wherein oxygen is taken up by the capillaries of the lung and carbon dioxide is released from the blood.

The rate, rhythm, and depth of respiration are reliable indicators of respiratory function and correlate well with a number of other systemic diseases and conditions (Table 3.6). Consequently, the rate, rhythm, and depth of respiration should be recorded on all new patients at the time of initial appointment and at all subsequent appointments on all patients with a history of cardiovascular and respiratory abnormalities.

Respiration in a male is primarily diaphragmatic and a noticeable use of the chest muscles in a male indicates air hunger (dyspnea, shortness of breath). Respiration in females is primarily coastal and the pronounced use of the diaphragm by a female indicates air hunger. The use of the accessory muscles of respiration (neck, shoulders) indicates air hunger, as seen in cases of congestive heart failure, bronchial asthma, or advanced pulmonary emphysema.

The normal respiration rate in adults is 16–20 breaths per minute and 24–28 breaths per minute in children. The rate typically increases by 4 breaths per minute for each Fahrenheit degree of body temperature elevation.

Technique

With the patient at rest, respiration is assessed by observing the rise and fall of the chest or upper abdomen over 1 minute. Patients may control respiration to some extent, so it is best determined without the patient's knowledge (awareness).

Temperature

Fever was a well-recognized sign of disease for centuries before the introduction of the thermometer. Now it is also well established that humans have a circadian temperature

Table 3.7. Conditions affecting body temperature.

Hypothermia	Hyperthermia
Anemia	Exercise
Alcoholism	Infection
Chronic debilitating disease	Ovulation
Hypothyroidism	Hyperthyroidism
Malignant hypothermia	Factitious fever

rhythm and this rhythm is difficult to disturb.

An awareness of this consistency assists clinicians with identifying disease or even factitious or self-induced illness (Table 3.7). Consequently, to establish a baseline value, the temperature of each patient should be recorded at the initial appointment and at all subsequent appointments when a patient presents with ominous signs of a serious odontogenic infection such as swelling (impending airway compromise, marked trismus), lymphadenopathy, chills, rapid respiration, hypotension, and tachycardia.

Factitious fever is an elevated temperature caused by some unnatural process (e.g., sipping a hot beverage right before having the temperature taken) that doesn't correlate with other presenting signs and symptoms. If a factitious fever is suspected, the patient should be closely observed while the temperature is retaken with a different thermometer. A factitious fever may be caused by a patient's desire to attract attention, gain sympathy, avoid work, or obtain a prescription for narcotics.

In health, the normal body temperature is about 37.0 °C. The maximum circadian variation is about 0.6 °C. Fever is defined as elevated body temperature of more than 38.2 °C rectally or 37.8 °C orally.

Technique

Prior to taking the temperature, the patient should be well rested and the medical history should have elicited information regarding the use of antipyretics that may have been taken within the past few hours.

Fever is most accurately diagnosed by rectal measurement; however, the technique is inappropriate in an oral healthcare setting. Oral measurements, which are normally about 0.6 °C lower than rectal measurements, are reproducible and convenient. Prior to determining the oral temperature, patients should not have smoked nor have had any hot or cold food or drink for at least 10 minutes. Other factors that may affect oral temperature include inadequate time of measurement (several minutes are required), mouth breathing, and hyperventilation. Measurement by tympanic membrane is the least accurate method of determining temperature.

Conclusion

Basic diagnostic procedures during initial and subsequent periodic limited physical examination of patients are of significant value in corroborating historical findings, formulating differential diagnoses, and determining the need for further diagnostic testing. Each procedure should be performed deliberately and systematically to permit the clinician maximum opportunity to detect and identify irregularities and abnormalities.

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4 Examination of the Head and Neck

A thorough examination of the head and neck is an essential component of the diagnostic process. Clinicians should be able to recognize normal variations and abnormalities. For maximum yield, the patient should be seated at eye level with the examiner. When an abnormality is noted, the following questions should be asked: (1) is the abnormality itself important, or is it an inconsequential finding; (2) could the abnormality be related to a potential intraoral finding; and (3) is the abnormality suggestive of an underlying systemic disorder?

Examine the Head and Face

Note the Position and Observe Movement of the Head at Rest

Tilting of the head may indicate an attempt to compensate for defective vision or hearing or to minimize discomfort in the neck. An exaggerated forward thrust of the head may be associated with an abnormality of the cervical vertebrae. Sudden, unexpected movements of the head with facial grimaces may simply be a habitual spasm. A to-and-fro bobbing of the head may be secondary to aortic insufficiency. A subtle but steady rhythmic tremor of the head may suggest **Parkinson's disease**. Constant licking of the corners of the mouth or smacking of the tongue is suggestive of **tardive dyskinesia**, a condition associated with neuroleptic drug therapy.

Parkinson's Disease

Parkinson's disease (PD) is a degenerative central nervous system (CNS) disorder, which may be either primary or secondary. Primary PD is characterized by degeneration of dopaminergic neurons of the substantia nigra and other brainstem dopaminergic cell groups. The loss of substantia nigra neurons results in depletion of the neurotransmitter dopamine. Secondary PD is associated with loss of, or interference with, the action of dopamine in the basal ganglia due to other degenerative CNS diseases, exogenous toxins, or drugs. It is estimated that at least half a million people in the United States suffer from PD. Most cases begin after the age of 50. Among individuals older than 65 years of age, reported estimates range from 1.5% to 3.0%. When PD begins at or before the age of 50, genetic factors appear to predominate. In late-onset PD, the triggers are primarily environmental.

Clinical Features

Regardless of its etiology, striatal dopamine deficiency leads to an impaired ability to control the smooth movement of skeletal muscle. The most common initial symptom is resting tremor of the hands. The classic "pill-rolling" motion of the fingers is often unilateral, usually decreases with voluntary movement, is absent during sleep, and may be exacerbated during emotional stress and fatigue. The hands, arms, and legs are affected in decreasing order of frequency. Other characteristic findings include rigidity, bradykinesia, and postural instability.

As PD progresses, the face becomes mask-like, the mouth remains open, and blinking is diminished. The patient may experience difficulty swallowing and tends to drool. Speech becomes slurred, monotonous, stuttering, and soft (hypophonia). Cognitive and emotional symptoms of PD include depression, memory impairment, and an inability to make executive decisions. Approximately 25% of patients with PD meet the criteria for dementia.

Diagnosis

The diagnosis of PD is suggested by a unilateral hand tremor, infrequent blinking, lack of facial expression, decreased movement, impaired postural reflexes, and a characteristic gait. The strongest support for the diagnosis of PD is provided by a positive clinical response to carbidopa and levodopa.

Treatment

Treatment protocols focus on increasing dopamine availability in the CNS and/or inhibiting the effects of acetylcholine. Levodopa, combined with carbidopa, remains the most effective symptomatic treatment for PD. Dental management of the patient with PD requires an awareness and accommodation of its progressive nature, which leads to physical, cognitive, and behavioral changes.

Tardive Dyskinesia

Tardive dyskinesia (TD) is a potentially irreversible, involuntary hyperkinetic disorder. The etiology of TD is not completely understood. One hypothesis suggests the possibility of dopamine receptor supersensitivity, while another contends that it is due to gamma-aminobutyric acid insufficiency. Advancing age is the most consistently established risk factor for TD, and there appears to be a linear correlation between age and both the prevalence and the severity of TD. Some ethnic differences, with higher rates in African Americans and lower rates in Chinese and other Asian populations, have been reported. Patients with diabetes mellitus are at a greater risk for developing TD, as are alcohol or drug abusers.

Numerous drugs have been implicated in TD. Prolonged treatment with neuroleptics such as risperidone, olanzapine, and haloperidol increase dopamine metabolism, which in turn results in increased free radical production. It is postulated that free radicals and other toxic agents damage the basal ganglia. A type of delayed-onset TD has also been reported in patients taking dopamine antagonists (e.g., metoclopramide, prochlorperazine), L-dopa, and amphetamines. Other drugs reported to be associated with acute TD include anticonvulsants (phenytoin, carbamazepine), oral contraceptives, chloroquine-based antimalarials, lithium, and tricyclic antidepressants. In patients taking these latter drugs, the symptoms of TD are reversible upon dose reduction or discontinuation of the offending drug.

Clinical Features

TD is characterized by rapid, jerky, writhing (twisting and turning), involuntary movements that most often affect the oro-facial region along with distorted tonicity of arm, leg, and trunk muscles. Involvement of the arm, leg, and trunk muscles occurs chiefly during walking. The movements are highly complex and appear to be well coordinated, but are senseless. Dyskinetic blinking is an early sign. Other movements commonly involve the orobuccal, lingual, and facial muscles, especially in older individuals. Puckering or pouting, lip smacking, chewing, jaw clenching or mouth opening, facial grimacing, and blowing are common features. As the condition progresses, writhing of the tongue is characterized by thrusting of the tongue out of the mouth as if “fly catching.” Repeated licking of the lips or pressing the tongue against the cheeks may produce an obvious bulge of the cheek.

The oro-facial musculature is involved in about 75% of affected patients. The limbs are involved in 50%, the trunk in up to 25%, and all three muscle groups are affected in

about 10% of patients. The movements of TD typically fluctuate in intensity over time, increase with emotional arousal, decrease with relaxation, and disappear during sleep. A majority of patients with TD have a mild disorder and may be unaware of its presence. However, 5–10% of patients suffer significant impairment. Oro-facial movements may lead to difficulty in eating or retaining dentures. Weight loss or cachexia may develop.

Diagnosis

The characteristic dyskinetic movements and their occurrence following exposure to neuroleptic drugs should suggest the presumptive diagnosis and mandate a medical consultation. A diagnosis is established following a complete neuropsychiatric evaluation.

Treatment

The best chance to induce remission, or at the least to minimize the progression of TD, is to discontinue the offending neuroleptic drug. In moderate-to-severe TD, drug therapy to treat involuntary movements may be necessary.

Note the Color of the Face

Pallor may be seen in patients with anemia or edema, and as a transient phenomenon in association with vasopressor syncope. **Cyanosis** may be evident in patients who have a congenital heart defect, congestive heart failure, or chronic obstructive pulmonary disease. Increased concentration of bilirubin in the blood leads to **jaundice** of the skin, sclera of the eyes, and mucous membranes. It may be indicative of excessive red blood cell destruction, hepatitis, cirrhosis, hepatocellular carcinoma, cholelithiasis, or pancreatic cancer. **Port-wine nevi**, or capillary or cavernous malformations affecting the cutaneous/mucosal distribution of the trigeminal nerve, may represent a manifestation of Sturge-Weber angiomatosis or Klippel-Trenauny syndrome. **Deep bronzing** of the face may be associated with Addison's disease.

Pallor

Pallor refers to the abnormally pale appearance of the skin and/or mucous membranes and is a principal sign of anemia. Anemia reflects a reduction in the oxygen in blood due to an abnormality in the quantity or quality of red blood cells and is characterized by a reduction in hemoglobin concentration and the volume of erythrocytes in a unit volume of whole blood (hematocrit). Anemia may be due to (1) defective proliferation of red blood cells, (2) defective maturation of red blood cells, (3) increased destruction of red blood cells, or (4) acute or chronic blood loss.

Edema of the skin may also produce pallor. The structural elements of the integument are rendered more distant from each other as they are separated by fluid. Light penetrating the skin meets a diminished quantity of pigment, more rays are reflected, and the skin appears paler than normal.

A common cause of transient pallor is vasodepressor syncope due to the dilatation of resistance vessels. This form of fainting is likely to occur in the oral healthcare setting in response to sudden emotional stress brought on by pain, surgical manipulation, sight of blood, or heat. Cerebral blood flow becomes significantly reduced, precipitated by a generalized, progressive, autonomic (adrenergic followed by compensatory cholinergic) discharge.

Clinical Features

Pallor is a physical sign. While there is a wide variability in skin color among individuals due to the size of capillaries and their depth below the skin surface, the clinical appearance of the conjunctivae, lips, and oral mucosae, which demonstrate less of a variation in color, provide a more reliable indication of pallor (Figures 4.1a–4.1d). Similarly, the color of the nail bed and a well-delineated lunula are also good indicators of pallor.



Figures 4.1a–b. Pallor.

Diagnosis

The diagnosis of pallor is based on a careful inspection of the skin, conjunctivae, lips, oral mucosae, and nail beds. Establishing the etiology of persistent pallor is the responsibility of the patient's physician.

Treatment

An appreciation for the extent and duration of pallor, along with any associated symptoms, may lead to the establishment of a specific etiology followed by appropriate treatment that will correct both the clinical signs and the underlying cause.

Cyanosis

Cyanosis is a physical finding characterized by a bluish discoloration due to an excess amount of reduced hemoglobin in the subpapillary venous plexus. It may be peripheral

or central. Peripheral cyanosis may be seen following exposure to low ambient temperatures, anxiety-induced vasoconstriction, Raynaud's disease–associated vasospasm, other obstructive peripheral vascular abnormalities, and low cardiac output. Central cyanosis results from arterial hypoxemia and may result from serious cardiac and/or pulmonary abnormalities.

Clinical Features

Peripheral cyanosis tends to affect the upper and/or lower extremities, particularly the palms of the hands, the soles of the feet, and the nail beds. When cyanosis is caused by heart or lung disease, in addition to the extremities, it affects the "central" parts of the body, that is, the torso, face, lips, and oral mucosae (Figures 4.2a and 4.2b).

Diagnosis

Cyanosis is best recognized under natural, bright daylight in regions where the skin is thick, unpigmented, and flushed, such as the ear lobes, cutaneous surfaces of the lips, and the nail beds. Cyanosis is less apparent in the oral mucosae of patients with light complexion when compared to patients with dark skin. Overall, the visual perception of "blueness" varies greatly among clinicians and it is believed that most clinicians do not perceive cyanosis until the oxygen saturation has fallen to less than 80%.



a



b

Figures 4.2a and b. Cyanosis.

Treatment

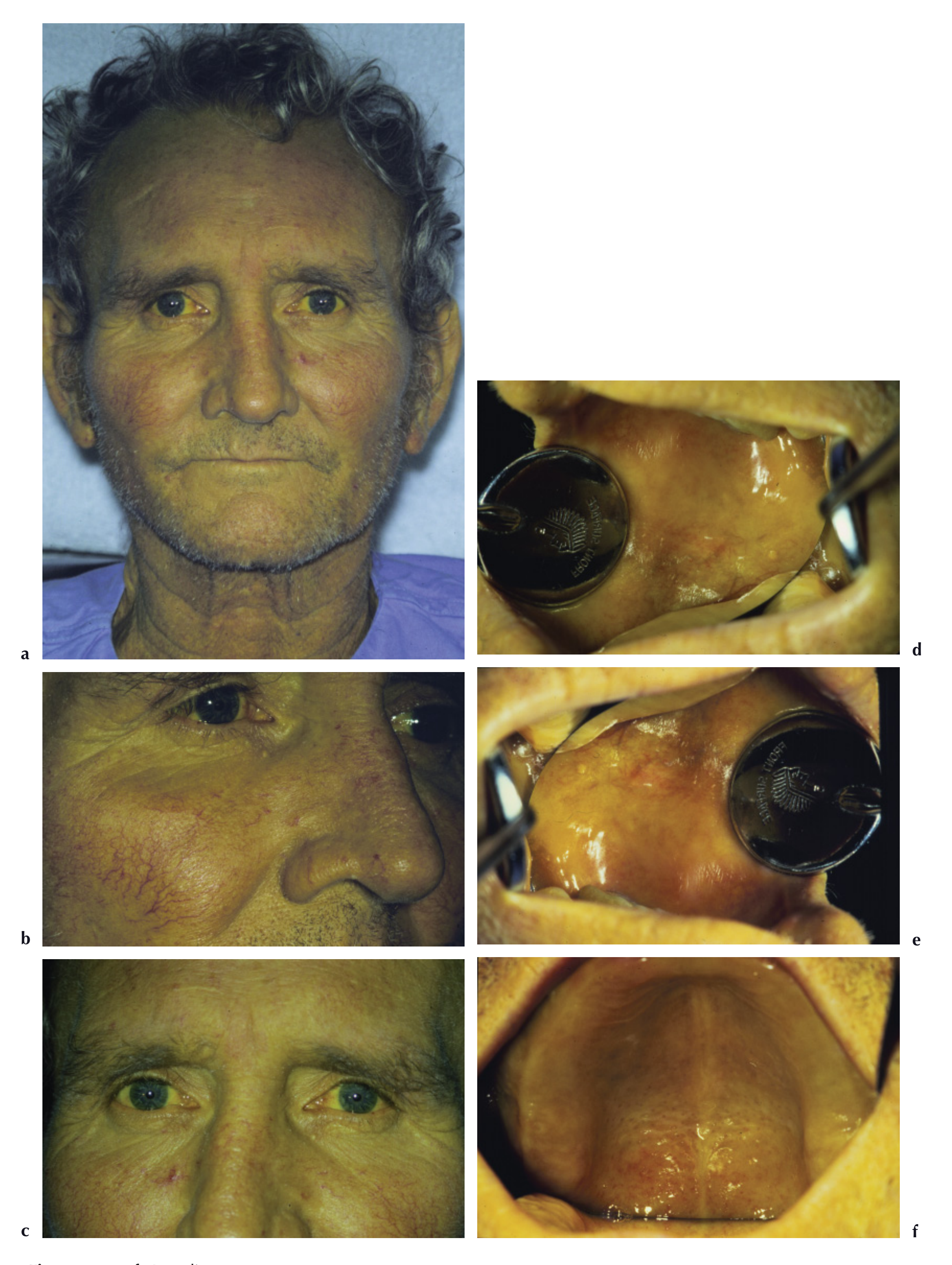
A careful history and physical examination by a physician are warranted to determine the specific etiology and, consequently, the most appropriate treatment.

Jaundice

Jaundice is the result of increased concentrations of bilirubin in blood. Red blood cells, when destroyed, release hemoglobin, and bilirubin is a by-product of subsequent hemoglobin catabolism. The bilirubin molecule is attached to albumin and is conjugated with glucuronic acid in the liver, rendering it water-soluble. Conjugated bilirubin becomes a constituent of the bile and is transported to the duodenum where it gives the fecal matter its characteristic color. The predominance of conjugated or unconjugated bilirubin can pinpoint the metabolic problem as prehepatic (destruction of red blood cells), hepatic (acute viral, drug-induced, or alcoholic hepatitis; subacute or chronic hepatitis; cirrhosis; hepatocellular carcinoma), or posthepatic (biliary tract obstruction by duct stones, duct stricture, or pancreatic cancer).

Clinical Features

Jaundice is a sign that typically affects the skin, oral mucosae, and sclera of the eyes (Figures 4.3a–4.3f). When associated with vascular spiders of the face, it may suggest acute or chronic hepatitis. Vascular spiders seldom occur in patients with jaundice resulting from obstruction secondary to bile duct stones or neoplasia.



Figures 4.3a–f. Jaundice.

Diagnosis

The diagnosis is based on visual inspection. While the skin is easily observed, the peripheral portion of the conjunctivae and the oral mucosae represent highly sensitive areas that may manifest jaundice before the skin. Medical consultation for further evaluation is warranted.

Treatment

The treatment of jaundice falls under the purview of the physician and is targeted at removing or controlling the underlying cause.

Port-Wine Nevi

A port-wine nevus is a characteristic form of a capillary hemangioma affecting the head and neck. While it often occurs as an isolated entity, its presence may be indicative of a more serious condition such as Sturge-Weber syndrome (encephalotrigeminal angiomatosis) or Klippel-Trenaunay syndrome.

Clinical Features

Port-wine nevi are purplish, diffuse macules with irregular borders that are sharply demarcated from the adjacent normal skin. They occur unilaterally on the face and follow the first, second, third, or all three divisions of the trigeminal nerve (Figures 4.4a–4.4e).

Diagnosis

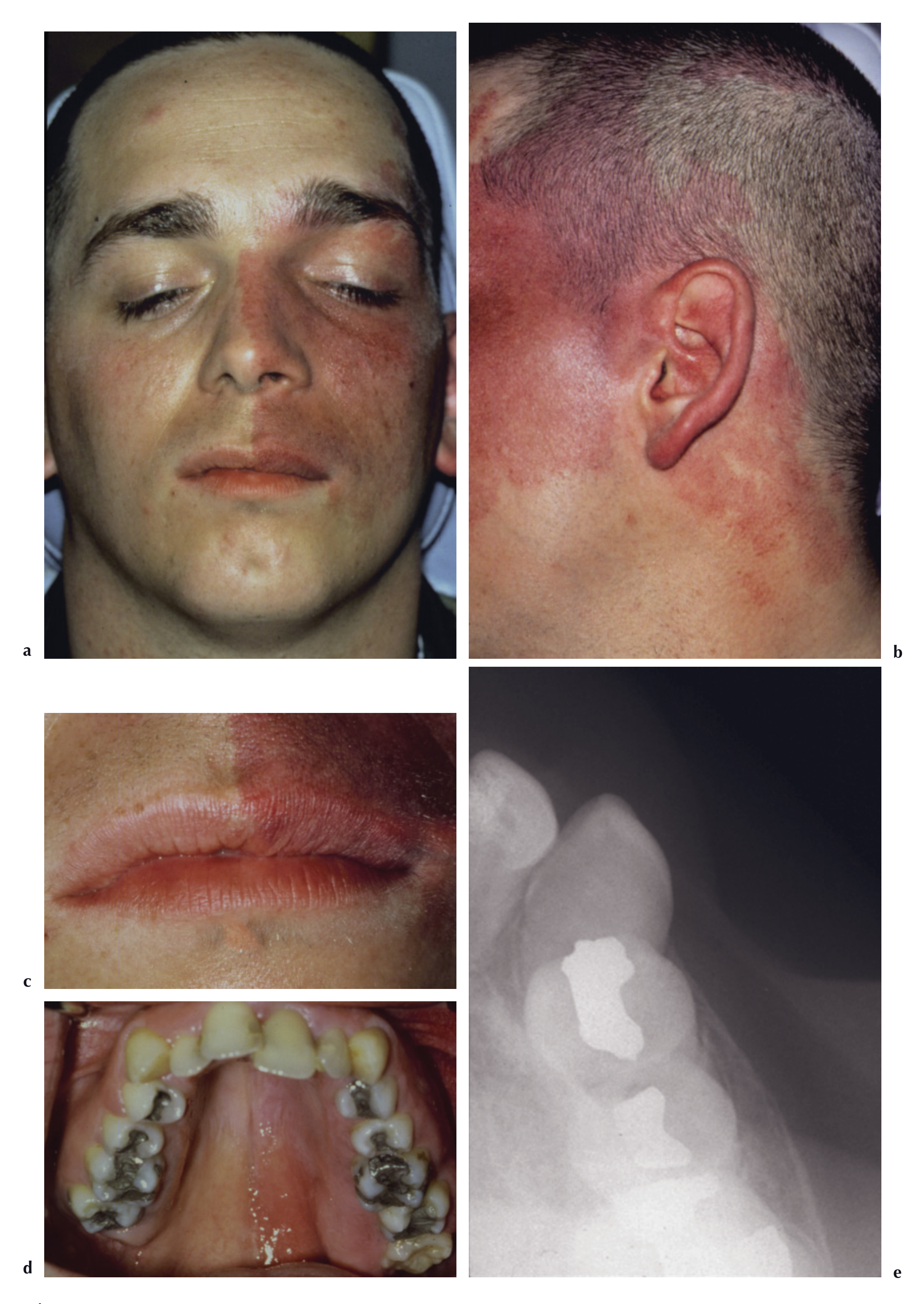
Port-wine nevi are easily diagnosed based on their characteristic appearance and history. A differential diagnosis should include Sturge-Weber syndrome and Klippel-Trenaunay syndrome (Table 4.1).

Treatment

Isolated port-wine nevi rarely require treatment and often involute during puberty. No consistently effective treatment is available to remove a port-wine nevus. Laser therapy may improve the cosmetic appearance of some lesions, but the improvement may only be temporary.

Table 4.1. Syndromes associated with port-wine nevi.

Sturge-Weber syndrome	Klippel-Ternaunay syndrome
Port-wine nevus	Port-wine nevus
Seizures	Venous malformation (varicosities)
Mental impairment	Bony and soft tissue hypertrophy
"Tramline" calcifications of the cerebral cortex	



Figures 4.4a–e. Port-wine nevi.

Deep Bronzing: Addison's Disease

Addison's disease (AD) is a potentially life-threatening endocrine disorder characterized by chronic adrenocortical hypofunction. The resultant lack of cortisol impairs the patient's ability to respond to physiologic stress. The cause of AD may be primary, secondary, or tertiary. Most cases of primary AD are caused by a gradual autoimmune destruction of the adrenal cortex. Less frequent causes of primary AD include glandular destruction by tuberculosis and human immunodeficiency virus (HIV) infection, histoplasmosis, primary or metastatic malignancies, amyloidosis, sarcoidosis, and drugs.

The incidence of primary AD in the general population is about 1/100,000. Secondary AD results from deficient adrenocorticotropic hormone (ACTH) secretion, usually due to the presence or treatment of a pituitary or hypothalamic tumor. Tertiary AD is the most frequently observed type and develops as a consequence of long-term high-dose therapeutic corticosteroid administration, which leads to suppression of the hypothalamic-pituitary-adrenal axis.

Clinical Features

The clinical manifestations of AD include fatigue, weakness, listlessness, orthostatic hypotension, weight loss, and anorexia. Patients may also report a craving for salt and experience periods of hypoglycemia. The distinguishing sign of AD is hyperpigmentation, or bronzing, most evident on sun-exposed areas of the skin, and patchy brown pigmentation of the buccal mucosae, tongue, and less frequently the lips and gingivae (Figures 4.5a–4.5c). The cause of these pigmentations is thought to be an increased compensatory pituitary synthesis of ACTH and melanin-stimulating hormone (MSH).



Figures 4.5a–c. Addison disease.

Diagnosis

The suspicion of AD is based on the presenting clinical signs and symptoms. Medical referral for further evaluation is warranted. Both passive (e.g., serum cortisol and ACTH levels) and provocative (e.g., insulin tolerance test) laboratory testing, combined with appropriate imaging tests, are required to establish the specific diagnosis.

Treatment

Both primary and secondary adrenocortical hypofunction require life-long glucocorticoid, and if necessary, mineralocorticoid replacement therapy. Therapy is empirically individualized to maintain normal weight and sense of well-being. Commonly prescribed glucocorticoid agents include hydrocortisone, prednisone, or dexamethasone. The chosen agent is often administered in the morning and evening to mimic the normal diurnal release of these hormones.

The impaired ability to respond to excess physiologic stress places a patient with AD at risk for developing an Addisonian crisis. During an Addisonian crisis, the patient becomes dangerously hypotensive and experiences hypoglycemia. Precipitating factors may include stress, infection, trauma, surgery, and the use of general anesthesia. Treatment of an Addisonian crisis is a medical emergency and includes prompt intravenous administration of saline and a glucocorticoid.

Observe Facial Characteristics

A massive face, craggy eyebrows, prominent nose, enlarged mandible, and macroglossia are characteristic of **acromegaly**. A puffy edematous face; dry, coarse, and scaly skin; and slowed speech and mentation are characteristic signs of **hypothyroidism**. A moon facies (fat deposition in the face), hirsutism, and acne may represent **Cushing's syndrome** associated with excess corticosteroids either from an endogenous or an exogenous source. **Bell's palsy**, characterized by unilateral paralysis of the facial muscles, may reflect transient or permanent seventh nerve damage.

Acromegaly

Acromegaly results from an overproduction of growth hormone after closure of the epiphyseal plates, and is usually due to an adenoma of the anterior lobe of the pituitary gland (Figure 4.6a). It is a rare condition with a prevalence of approximately 50–70 cases per million and an incidence of 3 cases per million per year. Acromegaly progresses slowly and insidiously and is most often diagnosed during the fourth decade of life, typically 10 years after initial onset.

Clinical Features

Clinical signs and symptoms include hyperhidrosis, muscle weakness, paresthesia, sleep apnea, hypertension, and heart disease. Skeletal growth can lead to prognathism, frontal bossing, nasal bone hypertrophy, and large hands and feet; coarsening of facial features, prominent lips, and macroglossia are common (Figures 4.6b–4.6g). An anterior open bite reflects supereruption of posterior and flaring of anterior teeth.

Diagnosis

The characteristic clinical presentation of acromegaly should alert the clinician and prompt a medical referral. Reviewing previous photographs of a patient may help to confirm clinical impression. Laboratory testing to establish the presence of elevated levels of growth hormone (GH) confirms the diagnosis. Radiographic imaging often reveals ballooning of the sella turcica, an indicator of pituitary enlargement. Other potential head and neck radiographic findings include enlargement of the paranasal sinuses, thickening of the outer table of the skull, increased condylar growth, and hypercementosis.



Figures 4.6a–g. Acromegaly.



Figures 4.6a–g. *Continued*

Treatment

Measures to abolish or obtund excess GH production include microsurgery, radiation therapy, and medical therapy combined with routine follow-up examinations. The skeletal and to some degree soft-tissue changes are permanent.

Hypothyroidism

Hypothyroidism is the most common form of thyroid dysfunction encountered in medical practice. If the condition occurs in infants and young children, it leads to cretinism. In adults it causes myxedema. Approximately 95% of patients with hypothyroidism have primary thyroid disease. Primary hypothyroidism may result from diseases or therapy that destroy thyroid tissue and include Hashimoto thyroiditis, radioactive iodine therapy for Graves' disease, subtotal thyroidectomy for Graves' disease or nodular goiter, deficient or excessive iodine intake, or subacute thyroiditis. Secondary hypothyroidism is due to inadequate production of thyroid-stimulating hormone (TSH) by the pituitary gland. Other infrequent causes of hypothyroidism include radiation to the neck, postpartum thyroiditis, and exposure to drugs such as iodine, lithium, and amiodarone.

Clinical Features

The thyroid gland may be normal, nonpalpable, or diffusely enlarged. The classic features of hypothyroidism include sinus bradycardia, hoarseness, nonpitting edema (myxedema) of the skin, periorbital edema, dryness of the skin, brittleness of the scalp hair, and an enlarged tongue and mouth breathing (Figure 4.7). Other signs and symptoms such as lethargy, depression, memory loss, cognitive impairment, modest weight gain, intolerance to cold, constipation, and neuromuscular dysfunction (vague aches and pains) are common.



Figure 4.7. Hypothyroidism.

Diagnosis

The diagnostic workup for a patient suspected of having hypothyroidism should include a thorough historical, clinical, and laboratory assessment. The laboratory hallmark of primary hypothyroidism is an increased compensatory serum TSH concentration and elevated cholesterol.

Treatment

The treatment of hypothyroidism involves lifelong thyroid hormone replacement, most commonly with levothyroxine. Once euthyroid state is attained, follow-up at regular intervals is recommended to avoid over-treatment (iatrogenic hyperthyroidism). Mild to moderate hypothyroidism is not a contraindication to dental care; patients with severe hypothyroidism have an impaired ability to respond to physiologic stress and may experience myxedema come. Patients with hypothyroidism, to a variable degree, are hyperreactive to CNS depressants.

Cushing's Syndrome

Chronic glucocorticoid excess leads to the development of Cushing's syndrome. Corticotropin-releasing hormone (CRH) synthesized in the hypothalamus stimulates pituitary ACTHf secretion. ACTH stimulates the adrenal cortex to release cortisol. Cortisol, the body's main glucocorticoid, exerts a negative feedback to help modulate further CRH and ACTH secretion. The pathogenic mechanisms of Cushing's syndrome can be divided into those that are ACTH-dependent and those that are ACTH-independent. A pituitary adenoma represents the most common type of ACTH-dependent Cushing's syndrome and accounts for 60–80% of all cases. The most common form of ACTH-independent Cushing's syndrome is due to adrenal hyperplasia. The estimated annual incidence of a pituitary adenoma is in the range of 0.1–1 new cases per 100,000.

Clinical Features

The clinical signs and symptoms of Cushing's syndrome include truncal obesity, glucose intolerance, muscle weakness, hypertension, osteoporosis, easy bruising, and edema (Figure 4.8a). Head and neck and oral manifestations of Cushing's syndrome include red cheeks, moon face, hirsutism, acne, and retarded dental age (Figures 4.8b and 4.8c).

Diagnosis

Patients presenting with signs and symptoms suggestive of Cushing's syndrome should be referred for medical evaluation. The most common test for screening for Cushing's syndrome is a 24-hour urine collection with analysis for urinary free cortisol excretion. More specific tests include the dexamethasone suppression test, ACTH assay, and CRH stimulation test. Imaging techniques (CT, MRI) are useful to localize pituitary or ectopic



Figures 4.8a–c. Cushing's syndrome.

ACTH-producing tumors and adrenal masses.

Treatment

Surgical treatment, depending on the etiology, may include trans-sphenoidal selective pituitary adenomectomy or bilateral adrenalectomy. Pituitary irradiation may be used in adult patients in whom a surgical approach is contraindicated. Depending on the extent of the therapeutic intervention, some patients will require lifelong hormonal replacement therapy. During periods of extreme stress (e. g., infection, surgery, and trauma), patients on replacement glucocorticoid therapy may require supplemental glucocorticoid administration to prevent adrenal insufficiency. Because of an increased susceptibility to infection and possible delayed wound healing, the postoperative status of surgical patients should be monitored closely.

Bell's Palsy

Bell's palsy (BP) is a common, acute, neurological disorder characterized by the sudden onset of facial nerve (C.N. VII) paralysis. The average annual incidence of BP is 13–34 patients per 100,000. It is more common in those aged 15–45 years, with the peak incidence in the third decade of life. The right and left sides are affected equally, and there is no gender predominance. Bilateral facial palsy is rare, with a frequency of less than 1%.

Proposed etiologies for BP include infectious, neoplastic, traumatic, and idiopathic causes. Edema and subsequent nerve entrapment of the facial nerve secondary to infection remains one of the most plausible causes. While BP is frequently attributed to a viral cause, no specific virus has been consistently demonstrated. Those most frequently implicated include herpes simplex virus type 1 (HSV-1) (Figure 4.9a), varicella-zoster virus (VZV), influenza type B virus, cytomegalovirus (CMV), adenovirus, mumps, coxsackie virus, Epstein-Barr virus (EBV), and HIV.

Clinical Features

The onset of BP is usually acute and peaks after a few days. The affected side of the face lacks expression during normal conversation. The inability to close the eyes places the patient at risk for corneal/conjunctival dryness and ulceration. The eyeball may be turned upward in an attempt to block entering light; other common findings include an inability to whistle, smile, or grimace (Figures 4.9b–4.9d). Hypersensitivity to sound (hyperacusis), loss of taste (ageusia), and pain near the mastoid area may also be present.

Diagnosis

The diagnosis of BP is relatively straightforward. However, a careful clinical history must be obtained to rule out other forms of facial nerve dysfunction such as trauma, otologic disease, and intracranial mass. A thorough examination of the head and cervical spine, including cranial nerve testing (i.e., eye closure, elevation of the eyebrows, smiling, frowning, and pursing of the lips) should be performed. Evidence of involvement of other cranial nerves should alert the clinician to consider other causes.

Treatment

The management of BP often requires a multidisciplinary approach. Eighty-three percent of all patients experience resolution with nothing more than supportive therapy. Recovery typically begins within 2 weeks but may take up to 6 months to complete. However, 17% of patients experience incomplete recovery and manifest moderate to severe residual impairment. The risk of incomplete recovery is increased by a multitude of factors including initial disease severity, older age (> 60 years), delayed onset of recovery (> 3 weeks), pregnancy, and the presence of other comorbidities such as diabetes mellitus and herpes zoster infection.

Supportive treatment includes a temporary patch to protect the exposed eye, ocular antibiotics and artificial tears to prevent corneal ulceration, and physical therapy to prevent contracture of the paralyzed muscles. Evidence for or against targeted drug therapy remains controversial, and further study is necessary. However, in a recent review, the use of a combination regimen of acyclovir and prednisone appeared to improve overall outcome, if commenced within 72 hours of the onset of symptoms.



Figures 4.9a–d. Bell's palsy.

Note Facial Architecture

Facial asymmetry may be caused by a **neurofibroma**, an **intramuscular hemangioma**, a **lymphangioma**, **fibrous dysplasia**, **progressive hemifacial atrophy**, an **osteosarcoma**, a **chondrosarcoma**, or by rare genetic disorders such as focal dermal hypoplasia. **Subcutaneous emphysema** may present as an acute, spontaneous form of facial asymmetry. A pronounced enlargement of the cranial vault is characteristic of **Paget's disease**. Multiple sebaceous or dermoid cysts and osteomas involving the skull may indicate **Gardner's syndrome**.

Neurofibroma

A neurofibroma is a benign tumor derived from the nerve sheath and is composed of a



Figures 4.10a–c. Neurofibroma.

proliferation of Schwann cells, perineural fibroblasts, and axons intermixed with mast cells. Most are discovered in adulthood and there is no gender predilection.

Clinical Features

Some neurofibromas are discrete and well circumscribed, whereas others are diffuse and infiltrating. Neurofibromas are commonly found on the skin. Intraorally, neurofibromas are most commonly found in the tongue, buccal mucosa, and lips; less commonly they may affect the alveolar bone (Figures 4.10a–4.10c). Neurofibromas arising in the tongue tend to be diffuse, while those arising in other locations tend to present as relatively well-demarcated, freely moveable, submucosal nodules. Depending on the degree of collagenization, neurofibromas may be soft or firm on palpation. Intraosseous neurofibromas typically present as a swelling without pain or parasthesia. Radiographically, the intraosseous neurofibroma is

relatively well demarcated, radiolucent, and may be either unilocular or multilocular. Root divergence may be evident.

Diagnosis

A definitive diagnosis of a neurofibroma is established by biopsy. The presence of more than two neurofibromas and six or more café-au-lait cutaneous macules should prompt the practitioner to consider the possibility of neurofibromatosis type 1 (von Recklinghausen's disease of the skin). It is a hereditary neurological disorder with an incidence of about 1 per 3,000. No racial or sex predilection is noted. Patients with neurofibromatosis type 1 are at an increased risk for developing cognitive deficits, epilepsy, and malignant nerve sheath tumors.

Treatment

There is no cure for neurofibromatosis. Treatment consists of routine monitoring and surgery when required to remove painful or disfiguring tumors. While the overall prognosis is good, malignant transformation of tumors occurs in 3–5% of the cases.

Intramuscular Hemangioma

Intramuscular hemangiomas (IMHs) are benign congenital neoplasms believed to evolve from an abnormally differentiated endothelial primordial network. This network is characterized by endothelial hyperplasia and malformations that enlarge by rapid cellular proliferation during the neonatal period, followed by a slow involution phase. Intramuscular hemangiomas account for less than 1% of all hemangiomas, of which approximately 15% occur in the head and neck area.

Clinical Features

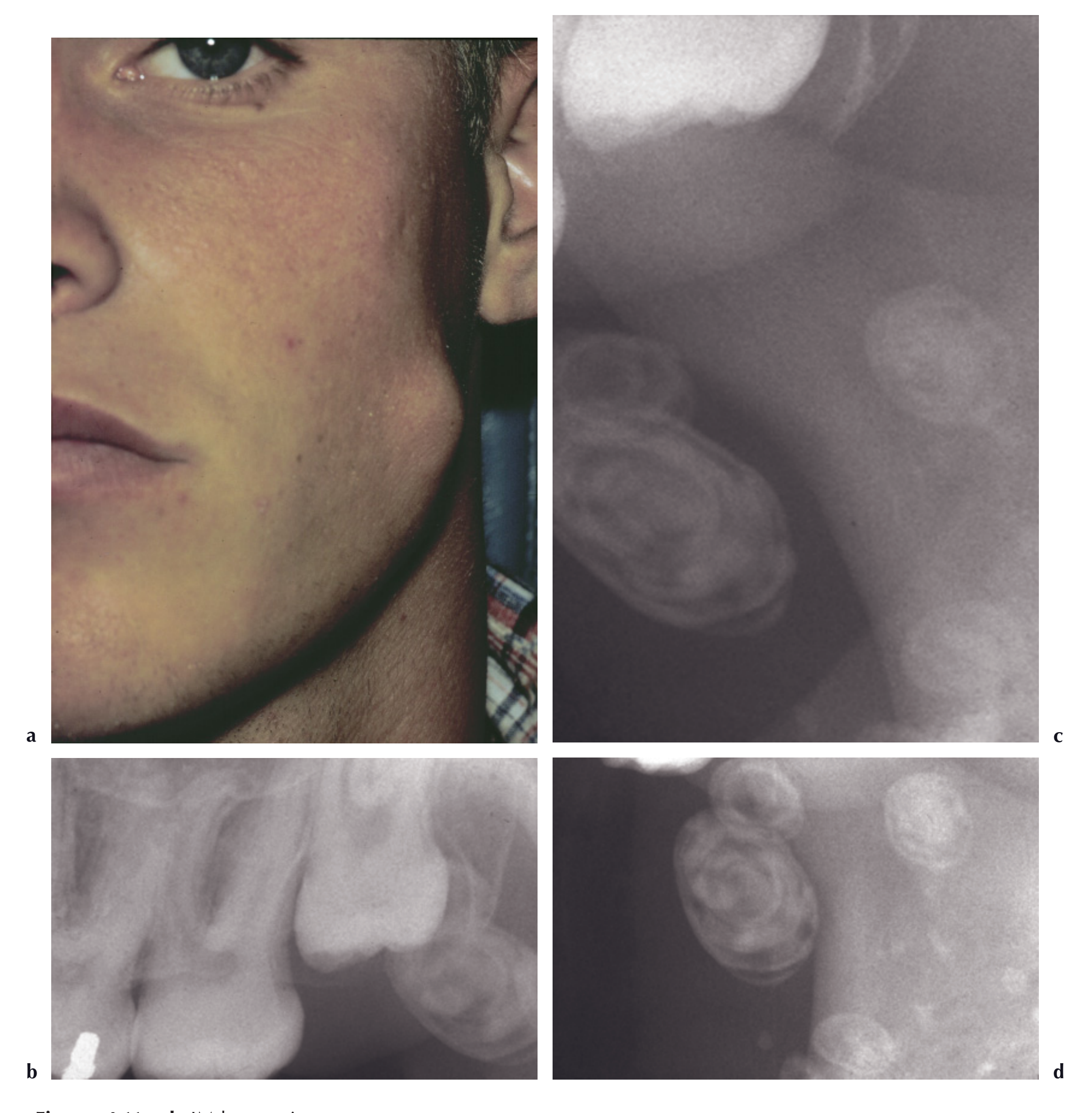
In the head and neck area, the masseter muscle represents the most common site (36%) for an IMH, followed by the trapezius, sternocleidomastoid, periorbital, and temporalis muscles. Intramuscular hemangiomas are usually asymptomatic until a growth spurt begins in the second or third decades of life, at which time pain occurs in about 50% of the cases. A palpable, fluctuant, or firm mass is present in up to 98% of the cases (Figure 4.11a). The size of an intramasseter hemangioma may increase with sustained masticatory pressure (clinching of teeth) and decrease subsequent to relaxation of the masticatory muscles. Radiographically, the presence of soft-tissue calcifications or phleboliths (spherical lamellar structures with irregular radiopaque and radiolucent areas) is seen in more than 15% of cases (Figures 4.11b–4.11d). Phleboliths are "stones" that appear to form within benign vascular lesions (hemangiomas, vascular malformations) secondary to a thrombotic episode.

Diagnosis

Sonography is the first-line imaging procedure for patients with soft-tissue swellings. Magnetic resonance imaging may be more reliable in detecting and delineating deeply situated and large IMHs. Angiography is diagnostic in most cases. The finding of a phlebolith is pathognomonic for a benign hemangioma. In many cases, a biopsy is required to confirm the diagnosis.

Treatment

Management of IMHs should be individualized according to the size and anatomic accessibility of the tumor, rate of growth, age of the patient, and cosmetic and functional considerations. If indicated, complete surgical excision is the treatment of choice. Local recurrence has been reported in about 18% of the cases.



Figures 4.11a–d. IM hemangioma.

Lymphangioma

Lymphangiomas are benign nonencapsulated tumors of lymphatic vessels. The majority of lymphatic malformations are present at birth and 80% become clinically evident before 2 years of age. However, lymphangiomas have been known to suddenly manifest in older children, adolescents, and adults. There is no gender predilection.

Clinical Features

Intraoral lymphangiomas most commonly occur on the tongue but are also seen on the palate, buccal mucosa, gingiva, and lips. Superficial microcystic lesions are manifested as papillary lesions, which may be of the same color as the surrounding mucosa, or may be slightly erythematous. Deeper macrocystic lesions appear as diffuse nodules or



Figures 4.12a–c. Lymphangioma.

masses without any significant change in surface texture or color. If the tongue is affected, considerable enlargement may occur. Facial lymphangiomas are the most common cause for enlarged lips (macrocheilia), enlarged ears (macrotia), and enlarged cheeks (macromala); cervicofacial lymphangiomas are associated with overgrowth of the mandibular body, which may lead to malocclusion (Figures 4.12a–4.12c).

Diagnosis

A lymphangioma should be considered in the differential diagnosis of all head and neck masses or swellings. Magnetic resonance imaging and a biopsy are often utilized to help confirm the diagnosis.

Treatment

Surgical excision is the treatment of choice since a lymphangioma is radioresistant and insensitive to sclerosing agents. Spontaneous regression is rare and lesions tend to recur after removal.

Fibrous Dysplasia

Fibrous dysplasia is a developmental abnormality of bone characterized by replacement of normal bone with fibrous connective tissue and woven bone trabeculae. Fibrous dysplasia represents 2% of all bone abnormalities, and there are two recognized variants: monostotic (single bone) and polyostotic (multiple bones).

The monostotic form is more common and accounts for approximately 80% of cases. It occurs primarily during the second decade of life with 75% of patients younger than 30 years of age at the time of diagnosis. The male-female ratio is approximately equal, and the maxilla is more commonly involved than the mandible.

There are two recognized variants of the polyostotic form. The Jaffe-Lichtenstein type is associated with "café-au-lait" dermal pigmentations (macules). McCune-Albright syndrome is characterized by polyostotic fibrous dysplasia, café-au-lait dermal macules, and precocious puberty or other endocrine disturbances arising in females.

Clinical Features

Fibrous dysplasia involving the bones of the craniofacial complex usually presents as a slowly progressive unilateral facial swelling (Figures 4.13a and 4.13b). Displacement of teeth may be present, along with abnormal occlusion due to jaw enlargement. Bone expansion typically causes buccal swelling as opposed to lingual swelling (Figure 4.13c). Maxillary lesions may extend into the sinuses, zygoma, sphenoid bone, and floor of the orbit.

Radiographically, the lesions appear radiolucent to radiopaque; the classic description is that of a smooth "ground-glass" appearance (Figure 4.13d). The lamina dura is often absent along with narrowing of the periodontal ligament space. Superior displacement of the mandibular canal strongly suggests fibrous dysplasia. Lesions may be unilocular or multilocular but generally are characterized by margins that blend into the surrounding normal bone.

Diagnosis

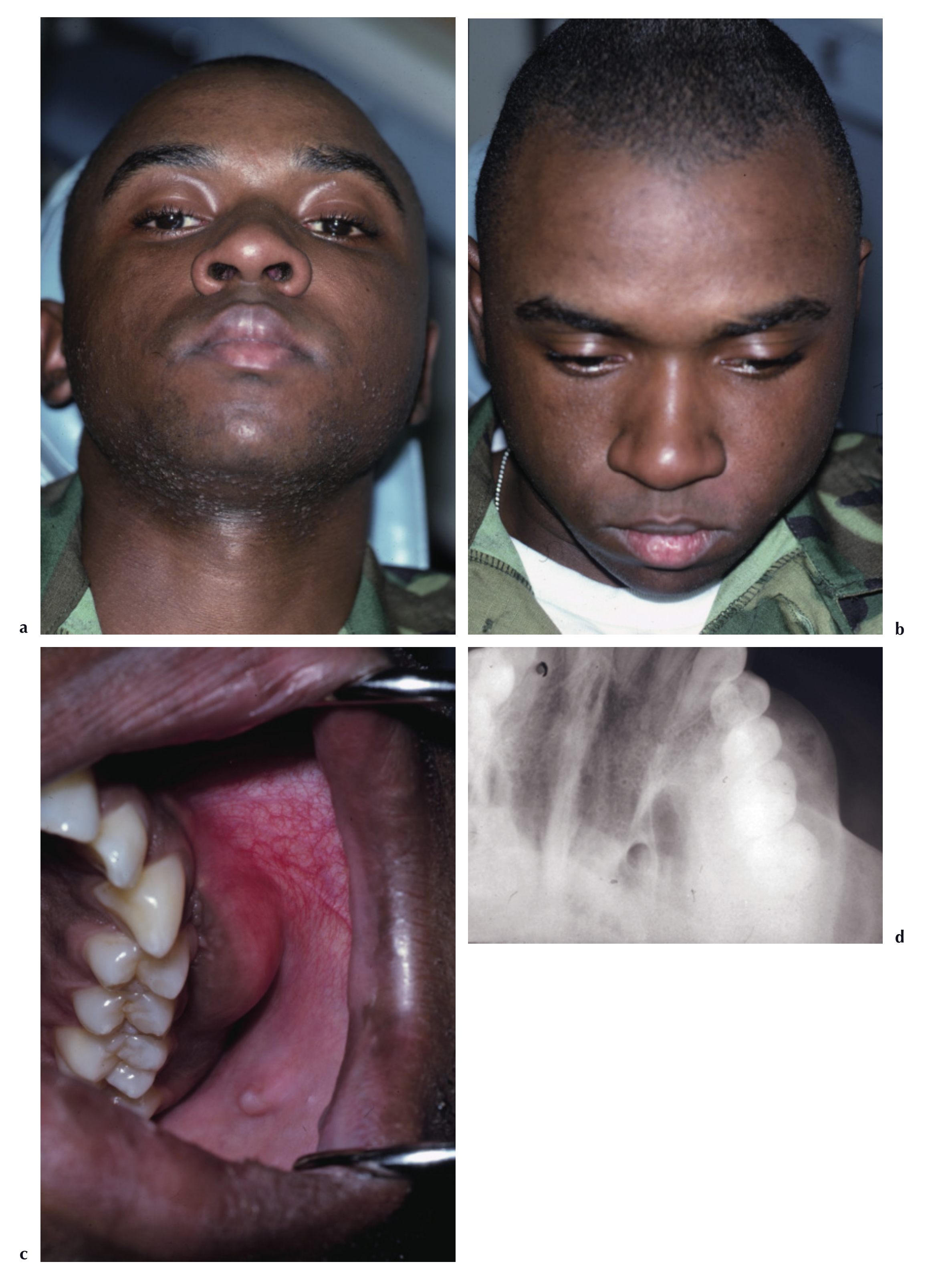
The diagnosis of fibrous dysplasia is based on correlating the characteristic histological findings with the clinical presentation and medical history. Histologically, fibrous connective tissue proliferation interspersed with trabecular woven bone has been described as resembling "Chinese characters." A primary consideration in the differential diagnosis is a central cemento-ossifying fibroma (Table 4.2).

Treatment

The treatment of fibrous dysplasia consists of conservative surgical resection since the condition tends to be self-limiting. Recontouring procedures generally are delayed until after puberty, when the lesions tend to become static. Radiation treatment is ineffective and

Table 4.2. Differential diagnosis of fibrous dysplasia and central cemento-ossifying fibroma.

Fibrous dysplasia	Central cemento-ossifying fibroma
< 30 years of age	Third and fourth decade
Maxilla or multiple bones	Body of mandible
Poorly defined, diffuse margins	Well demarcated
Ground-glass appearance	Nodular opacities



Figures 4.13a–d. Fibrous dysplasia.

may promote malignant transformation. The polyostotic form may actually undergo spontaneous malignant transformation without radiotherapy.

Progressive Hemifacial Atrophy

Progressive hemifacial atrophy (Parry-Romberg syndrome) is a rare pathologic process characterized by slowly progressive but self-limited unilateral atrophy of the face. It is thought to represent a focal or localized form of scleroderma. The etiology is unknown. Symptoms usually begin in the first or second decade of life, but onset has also occurred in patients older than 60 years. Progression of the disease tends to persist for 2–10 years, followed by a period of burn-out or stability.

Clinical Features

Hemifacial atrophy variably involves skin, subcutaneous tissues, fat, muscles, and less commonly the underlying osseous tissues along one or more of the trigeminal dermatomes. Many patients demonstrate normal neurological function, although symptoms attributable to ipsilateral cerebral hemispheric dysfunction are not uncommon. Neurological involvement may lead to seizures, migraine headaches, cranial nerve deficits, masticatory spasms, cognitive abnormalities, and fixed focal deficits.

Affected areas may demonstrate pigment changes, alopecia, and a change from normal hair color to white (Figures 4.14a–4.14c). Ophthalmic defects such as ipsilateral Horner syndrome, blepharophimosis, chronic cyclitis, iritis, cataract, and secondary glaucoma may occur. Oral manifestations may include hemiatrophy of the lips, tongue, jaws, and buccal fat pad. Corresponding radiographic changes may include hemispheric calcifications, leptomeningeal enhancement, loss of cortical gyration, increased density of deep white and gray matter structures, and atrophy.

Diagnosis

The diagnosis of progressive hemifacial atrophy is based on clinical presentation, neurological findings, and computed tomography and magnetic resonance imaging.

Treatment

There is no cure for this disease. Treatment is limited to surgical reconstruction of the affected areas.



Figures 4.14a–c. Hemifacial atrophy.

Osteosarcoma

Osteosarcoma is the most frequently encountered skeletal malignancy, accounting for about 35% of all cases. It is a true malignant neoplasm of bone, which may occur as a central, juxtacortical, or peripheral lesion. Primary osteosarcoma of the jaws is uncommon, accounting for about 10% of all osteosarcomas reported. It is less aggressive and less likely to metastasize than osteosarcoma arising in long bones. Predisposing etiologic factors may include a history of fibrous dysplasia, Paget's disease, radiation therapy, or trauma to the bone. The mean age at presentation in the maxilla/mandible is 30–34 years (10 years younger for long bones), with men affected twice as often as women. The maxilla and mandible are affected about equally.

Clinical Features

A patient typically presents with loose teeth in the area of the tumor, localized swelling, and, at times, significant facial asymmetry (Figure 4.15a). The overlying mucosa is



Figures 4.15a–d. Osteosarcoma.

usually normal in appearance, but may be red with small telangiectatic surface vessels. Paresthesia and/or pain are late features of the disease, as are nasal obstruction, blurred vision, and headache.

Radiographically, the lesions may appear radiolucent, radiopaque, or mixed. Mixed lesions exhibit the characteristic lamellar pattern of ossification, classically described as a "sunburst" or a "sunray" (Figures 4.15b–4.15d). Other characteristic radiographic signs include a uniform widening of the periodontal ligament space and an increase in height of the alveolar process when compared to unaffected areas. Periosteal reactions may include the deposition of irregular new bone or lamellar new bone (onion skin).

Diagnosis
The diagnosis of osteosarcoma is established by a biopsy.

Treatment
Most cases of osteosarcoma are treated with chemotherapy (neoadjuvant) before radical surgical resection, at times followed by adjuvant chemotherapy. With early detection and treatment, longtime survival rates can be as high as 60%.

Chondrosarcoma

Chondrosarcoma is a malignant cartilagenous tumor. It accounts for approximately 11% of all skeletal malignancies, with only about 1–2% of cases occurring in the head and neck area. The median age at the time of diagnosis for those tumors arising in the head and neck area is 52 years, and there is a slight male predominance. The etiology is unknown. It has been suggested that chondrosarcoma is more likely to develop in individuals who have a specific pre-existing genetic condition, such as Ollier's disease, Maffucci syndrome, and multiple hereditary exostoses or osteochondromatoses. People affected by these conditions are more susceptible because the pre-existing benign bone tumors have the potential to undergo malignant degeneration. Adults with Paget's disease may be at increased risk for osteosarcoma or chondrosarcoma.

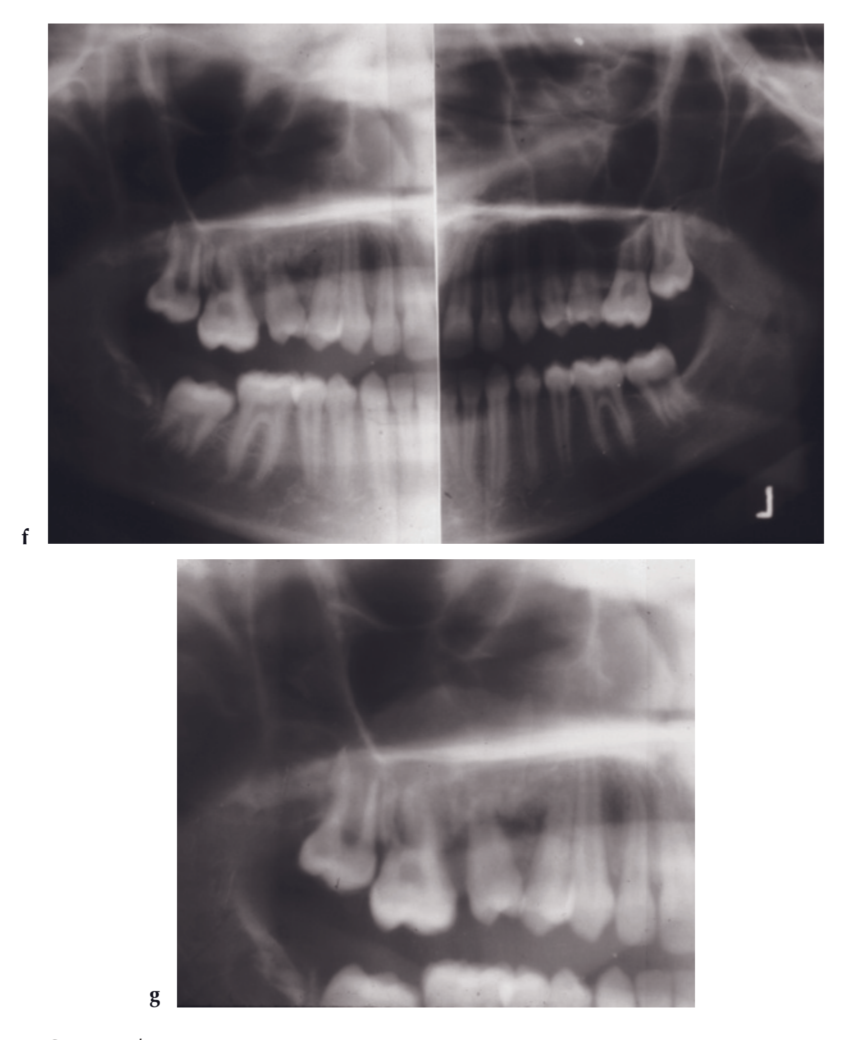
Clinical Features
Chondrosarcomas are more common in the maxilla than in the mandible. Although some tumors may follow an aggressive clinical course, chondrosarcoma is usually slow-growing and may lead to subtle facial asymmetry (Figure 4.16a). Symptoms vary depending on the location and size of the tumor. In the oral cavity, chondrosarcoma most commonly presents as a painless swelling or mass leading to tooth separation and mobility (Figures 4.16b–4.16d). Radiographically, lesions in the jaws may vary from radiolucent to mixed radiolucent/radiopaque, while a tumor that invades the sinuses usually appears as radiopaque (Figures 4.16e–4.16g).

Diagnosis
The diagnosis of chondrosarcoma is established by a biopsy.

Treatment
The most common therapeutic approach is surgical excision with or without radiotherapy. The overall 5-year survival rate for chondrosarcoma affecting the head and neck region is about 87%.



Figures 4.16a–g. Chondrosarcoma.



Figures 4.16a–g. *Continued*

Subcutaneous Emphysema

Subcutaneous emphysema of the head and neck region and thorax is caused by the introduction of air into fascial planes. Because of the relative looseness of mucosal connective tissue, air can accumulate in these areas as a result of increased intraoral pressure associated with coughing, sneezing, blowing the nose, rinsing the mouth, playing a musical instrument, air-generating dental instruments (dental handpiece or an air-water syringe), and the release of oxygen from hydrogen peroxide. This can create spaces of considerable size. Along with air, the introduction of bacteria and foreign bodies into the fascial planes can lead to infection.

Clinical Features

A sudden onset of facial swelling with a sensation of fullness of the face may be the first clinical sign of subcutaneous emphysema (Figures 4.17a–4.17c, 4.18a, and 4.18b). The patient may also complain of intraoperative tingling in the periorbital area, which may lead to closure of the eyelids on the affected side. Crepitation, pain, and tenderness may



Figures 4.17a–c. Subcutaneous emphysema.

be noted when palpating the swelling. Radiographically, subcutaneous emphysema may appear as a large multilocular soft-tissue radiolucency displacing the posterior pharyngeal wall forward. Chest x-rays and soft-tissue films of the neck may display interstitial air emphysema of the retropharynx on the involved side of the face and neck.

Diagnosis

Early recognition is the most important aspect in the diagnosis of subcutaneous emphysema. Crepitation upon auscultation with a stethoscope is almost pathognomonic for subcutaneous emphysema. A sudden swelling of the neck, difficulty breathing, a brassy quality to the voice, and crackling when the swollen region is palpated are char-


a


b

Figures 4.18a and b. Subcutaneous emphysema.

acteristic features of mediastinal emphysema. A crunching noise may be heard on auscultation, and air spaces may be noted in anteroposterior and lateral chest radiographs. An allergic reaction, a hematoma, angioedema, and an esophageal rupture should be considered in the differential diagnosis.

Treatment

The treatment of subcutaneous emphysema is mainly supportive. Broad-spectrum antibiotics should be prescribed for 10 days to prevent secondary infection. Cough suppressants may be prescribed to prevent further entry of air into the fascial planes. A follow-up appointment within 48 hours is imperative to monitor for resolution and signs of infection. Severe cases mandate medical referral. Serious complications include meningitis when the maxilla is involved and mediastinitis when the mandible is affected. In severe cases, a pneumothorax or air embolism may occur.

Paget's Disease

Paget's disease (osteitis deformans) is a heterogenous, progressive bone disease characterized by brisk bone remodeling. The risk of developing Paget's disease increases with age, affecting an estimated 1% of those over the age of 40 years in the United States. Men are at slightly greater risk than women. The etiology is unknown, but autoimmunity and a variety of environmental factors have been postulated as contributing to the disease.

Clinical Features

Paget's disease may affect one (monostotic) or several (polyostotic) bones, and most cases are asymptomatic and discovered incidentally. The most commonly affected sites include the pelvis, skull, vertebra, femur, and tibia. Common clinical signs and symptoms include osseous distortion or expansion and mild-to-moderate bone pain. Skull involvement is estimated to occur in about 27% of cases and may result in hearing loss and vestibular problems in up to 89% of those affected. While osteosarcoma is a rare complication, most cases of adult osteosarcoma occur in patients with Paget's disease.

The characteristic radiographic findings of Paget's disease include lytic changes, characterized by osteoporosis; and sclerotic changes, characterized by a cotton-wool appearance. An overall mosaic of lytic and sclerotic findings is frequently noted. Numerous dental abnormalities, such as malocclusion,

hypercementosis, tooth mobility, root resorption, pulp calcification, osteomyelitis, poor fitting prostheses, and excessive postsurgical bleeding have been associated with Paget's disease.

Diagnosis
The diagnosis of Paget's disease is confirmed by correlating the presenting signs and symptoms with appropriate laboratory tests. The characteristic laboratory finding is an elevated serum alkaline phosphatase. A bone scan is useful to determine the extent of bone involvement.

Treatment
Therapy is dictated by the extent of the disease and associated symptoms. Bisphosphonates are powerful inhibitors of bone resorption and have been shown to be highly effective in stabilizing bone turnover and reducing disease-related symptoms. However, these drugs are not curative.

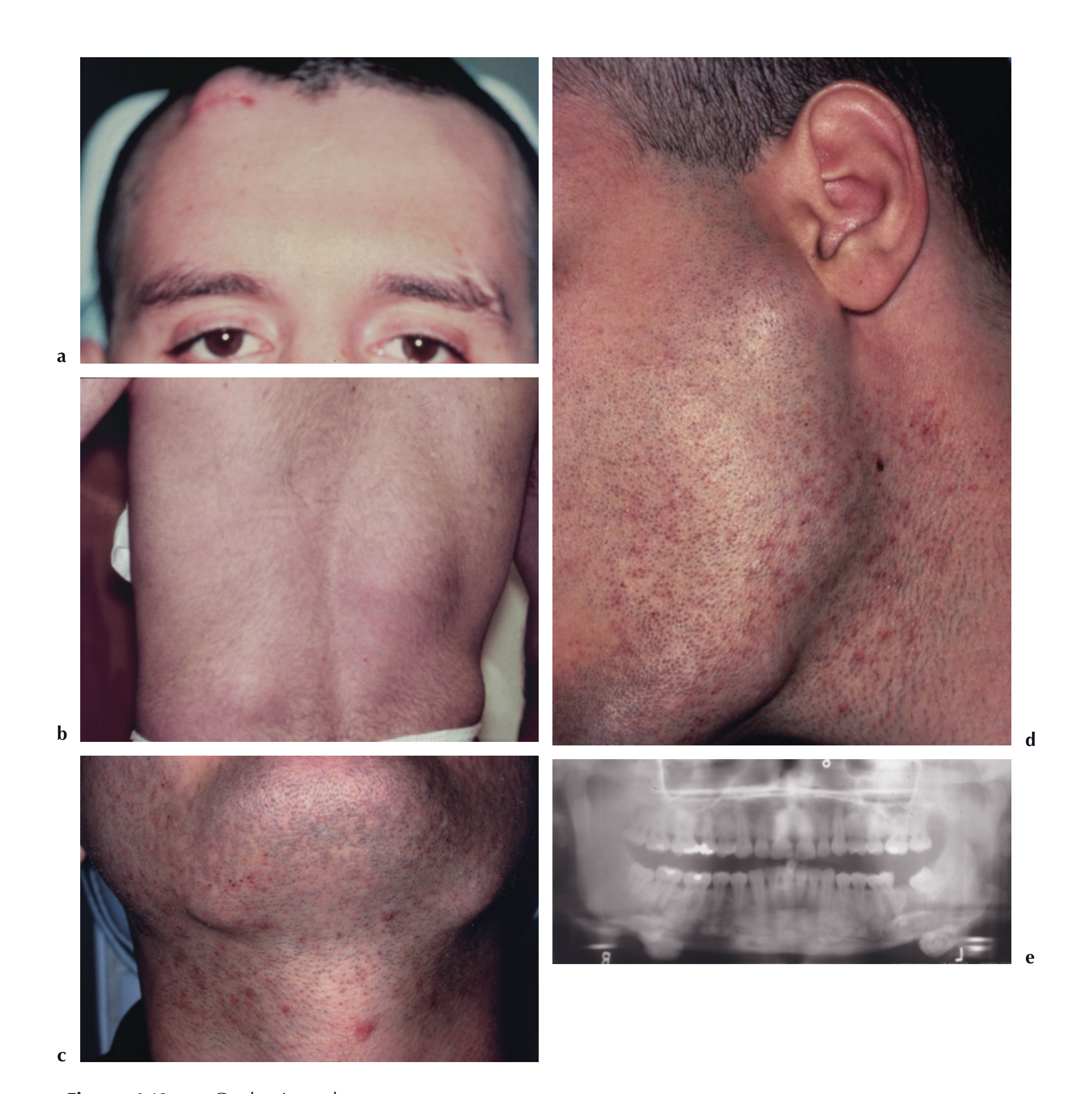
Gardner's Syndrome

Gardner's syndrome is a variant of familial adenomatous polyposis. It is a hereditary autosomal dominant disorder characterized by polyps in the gastrointestinal tract (stomach, small intestine, and large intestine), bone tumors, and skin and soft-tissue tumors. These polyps have a 100% chance of transforming to malignancy. Recently, the extracolonic manifestations were expanded to include retinal lesions, dental anomalies, and various cancers. Although the reported incidence of familial adenomatous polyposis is 1 in 8,000, Gardner's syndrome is much less common, affecting about one individual per million in the general population.

Clinical Features
Epidermoid cysts, which develop before puberty, are present in approximately 50–65% of patients (Figures 4.19a and 4.19b). Other commonly observed cutaneous neoplasms include fibromas, lipomas, leiomyomas, neurofibromas, and pigmented skin lesions. Desmoid tumors and multifocal, pigmented lesions of the ocular fundus have been described. Osteomas are an essential component of Gardner's syndrome, and the mandible is the most common location (Figures 4.19c–4.19g). Other oral findings may include congenitally absent teeth, hypercementosis, dentigerous cysts, impacted teeth, supernumerary teeth, and fused or unusually long roots. Long bones may demonstrate cortical hyperostosis or the formation of osteomas. Osteomas generally precede the clinical and radiographic evidence of colonic polyps.

Diagnosis
The presence of the characteristic stigmata of Gardner's syndrome should prompt referral for a thorough medical evaluation. Panoramic radiographs are particularly useful to screen for Gardner's syndrome. The presence of osteomas is a reliable marker of Gardner's syndrome.

Treatment
Medical management of colonic polyps with tamoxifen and sulindac has been of benefit for some patients, but surgical intervention is typically required. Since siblings and children of patients with Gardner's syndrome have a 50% chance of being affected, all first-degree relatives must be screened for the disorder. Regular screening by a gastroenterologist is mandatory for all patients with Gardner's syndrome.



Figures 4.19a–g. Gardner's syndrome.


f


g

Figures 4.19a–g. *Continued*

Assess the Character and Integrity of the Skin

A variety of skin lesions may be noted on the face. Acne is a common finding in teenagers, and is characterized by multiple pustules and comedons. **Rosacea** is an acne-like condition that typically affects older individuals. **Psoriasis** is a common skin disorder that affects the eyebrows and anterior scalp and presents as scaly plaques. **Seborrheic keratosis** is a discrete, rough-surfaced, and brown to black papule that typically occurs in patients over 40 years of age. Important malignancies that commonly affect the face are **basal cell carcinoma**, squamous cell carcinoma, and melanoma. Basal cell carcinoma initially presents as a persistent papule with surface telangiectatic blood vessels and evolves into an umbilicated ulcer. Squamous cell carcinoma typically appears as a persistent, rapidly growing ulcer with raised borders. A melanoma may arise de novo or from a pre-existing nevus. Any nevus that demonstrates bleeding, growth, a change in color, or the development of satellite lesions should be biopsied in order to rule out potential malignant transformation to melanoma.

Rosacea

Rosacea is a chronic and progressive cutaneous vascular disorder affecting the nasal and malar areas of the face. It appears to be inflammatory in nature, but the specific etiology is unknown. There is a slight female predilection, and patients with fair complexion who flush or blush easily or have a family history of rosacea are most susceptible. Specific triggers such as sunlight or cosmetics may contribute to the disease process. Most patients are diagnosed between the ages of 30 and 60.

Clinical Features

Classic signs of rosacea include flushing, persistent erythema, papules, pustules, and telangiectasia (Figures 4.20a and 4.20b). Four subtypes of rosacea are recognized: erythematotelangiectatic, papulopustular, phymatous, and ocular. The phymatous subtype has a predilection for men and is characterized by rhinophyma, large lobulated masses affecting the tip and wings of the nose. The ocular subtype affects up to 58% of rosacea patients. Typical ocular signs and symptoms include blepharitis, dry eyes, burning, soreness, and




Figures 4.20a and b. Rosacea.

grittiness. In severe cases, corneal involvement may lead to blindness.

Diagnosis
When rosacea is suspected, the patient should be referred to a dermatologist for definitive diagnosis and subsequent management. Other conditions to consider in the differential include acne vulgaris, seborrheic dermatitis, chronic contact dermatitis, and lupus erythematosus.

Treatment
Therapeutic options for rosacea include topical and oral agents, laser and light treatments, and surgical interventions. Patients with rosacea should avoid known triggers such as sun exposure and any offending cosmetic products. The most commonly prescribed topical agents are metronidazole, azelaic acid, and sodium sulfacetamide-sulfur. The most commonly prescribed oral agent is low-dose doxycycline monohydrate. Surgical or laser therapy may be necessary to ablate cosmetically unacceptable rhinophyma.

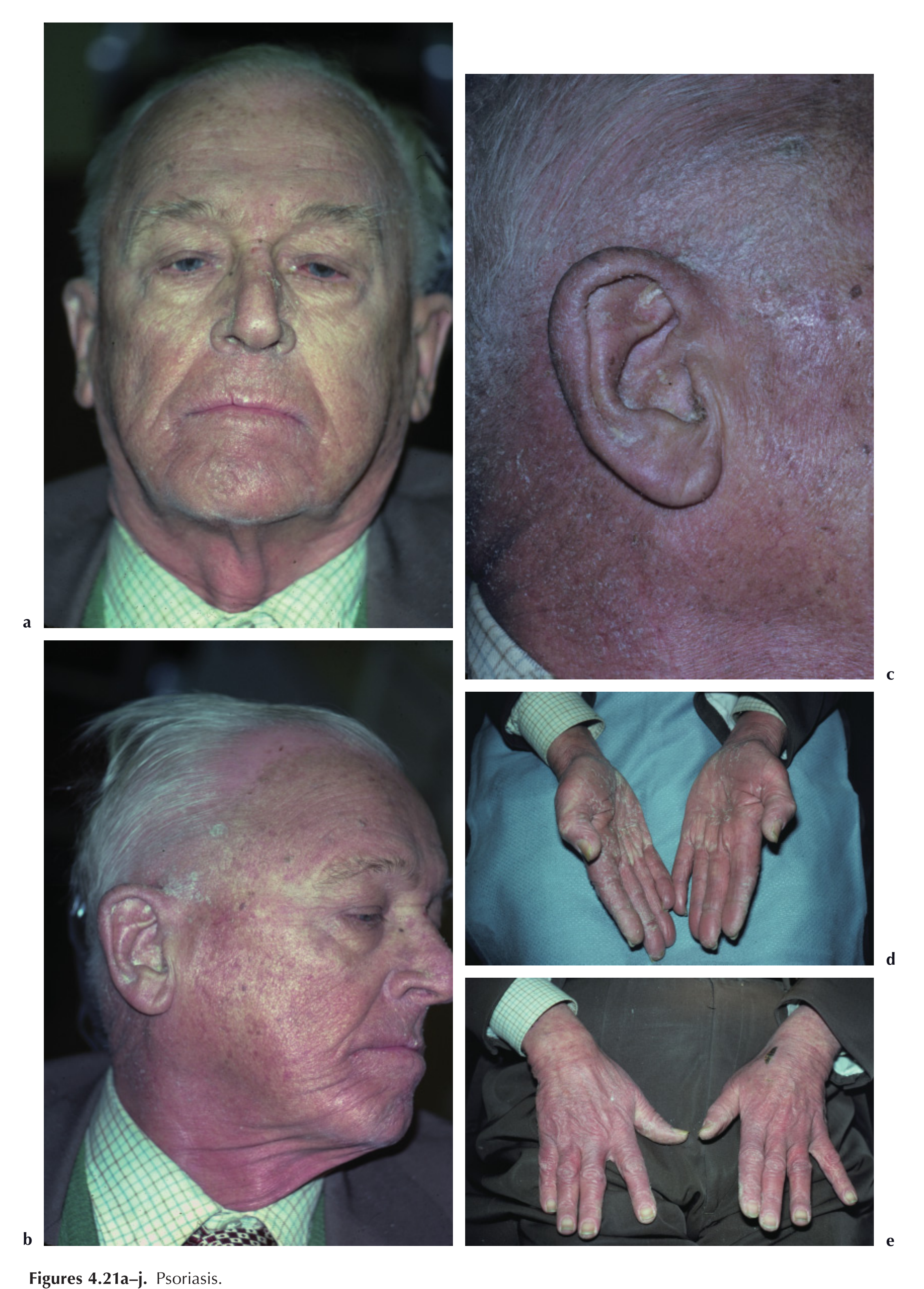
Psoriasis

Psoriasis is a common chronic, recurrent, inflammatory skin disorder. It is characterized by hyperproliferation of the epidermis and inflammation secondary to a T-cell (CD4)-mediated autoimmune process. The pool of proliferating keratinocytes is expanded, their migration from the basal cell layer to the surface is more rapid, and the cell cycle is shortened. Psoriasis affects 1–3% of the world's population. It affects all ages, but is primarily a disease of adults.

Clinical Features
The most commonly observed form of psoriasis is the plaque form, which presents as circumscribed, thickened, silvery, scaly papules and plaques that may be pruritic. They occur most often on the scalp, face, nails, eyebrows, elbows, knees, buttocks, and sites of local trauma (Koebner phenomenon) (Figures 4.21a–4.21j). Psoriatic arthritis is a related condition in which the joint tissues are targeted by the autoimmune process underlying psoriasis. Psoriasis is a dynamic disease that often appears to wax and wane over time. Numerous factors such as infection, stress, climate changes, and certain medications may contribute to disease flares.

Diagnosis
The diagnosis of psoriasis is based on clinical findings, which may be supplemented by a skin biopsy.

Treatment
At present, there is no cure for psoriasis. The extent of care is determined on a case-by-case



Figures 4.21a–j. Psoriasis.



Figures 4.21a–j. *Continued*

basis with the target of decreasing disease severity and improving the patient's quality of life. The number of therapeutic agents available to manage psoriasis is vast and can generally be divided into three groups: (1) topical agents, (2) light therapies, and (3) systemic agents. Newly developed biologic therapies that target specific cytokine networks in the disease process may prove useful.

Seborrheic Keratosis

Seborrheic keratosis is a benign skin growth surfaced by loose adherent scales. The etiology of seborrheic keratosis is unknown but has been suggested to include genetic predisposition, exposure to sunlight, human papillomavirus, and hyperplasia of melanocytes. It is not contagious.

Clinical Features

Seborrheic keratosis is commonly seen in Caucasians and rarely identified in African Americans and Native Americans. The lesions initially present as flat, well-demarcated brown macules that gradually become greasy papules with a verruca-like surface, which appears to be stuck to the skin. The look is often compared to brown candle wax that was dropped onto the skin. They are usually located on the face, body, and limbs.

Diagnosis

The diagnosis of seborrheic keratosis is primarily clinical. In some cases, differentiating between pigmented seborrheic keratosis, malignant melanoma, and other cutaneous lesions might be difficult. Such equivocal cases warrant a referral to a dermatologist for further evaluation. Any seborrheic keratosis that presents with an atypical appearance or undergoes dramatic change should be biopsied.

Treatment

The management of seborrheic keratosis is usually considered to be cosmetic. When necessary, most seborrheic keratoses are treated by one of three methods: (1) liquid nitrogen cryotherapy, (2) curettage or excision, and (3) electrosurgery.

Basal Cell Carcinoma

Basal cell carcinoma (BCC) is the most common skin cancer. Although metastasis rarely occurs, it is locally invasive. BCC accounts for 75–80% of all skin cancers in whites and an estimated 850,000 new cases occur each year in the United States. Despite its prevalence in adults, it is extremely rare in children. The primary established risk factor for BCC is ultraviolet radiation, and fair-complected individuals are at highest risk for developing this neoplasm.

Clinical Features

BCC is fairly typical in appearance and can usually be diagnosed by an experienced observer on clinical grounds alone. The classic form presents as an indurated nodule with a rolled edge, often associated with surface telangiectasias or a pearly appearance (Figures 4.22a, 4.22b, 4.23, and 4.24). Some tumors may ulcerate (Figures 4.25 and 4.26). Basal cell carcinoma in children has been associated with familial syndromes such as


a


b

Figures 4.22a and b. BCC.

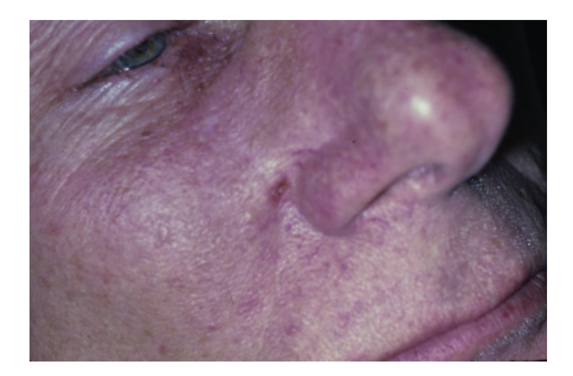


Figure 4.23. BCC.

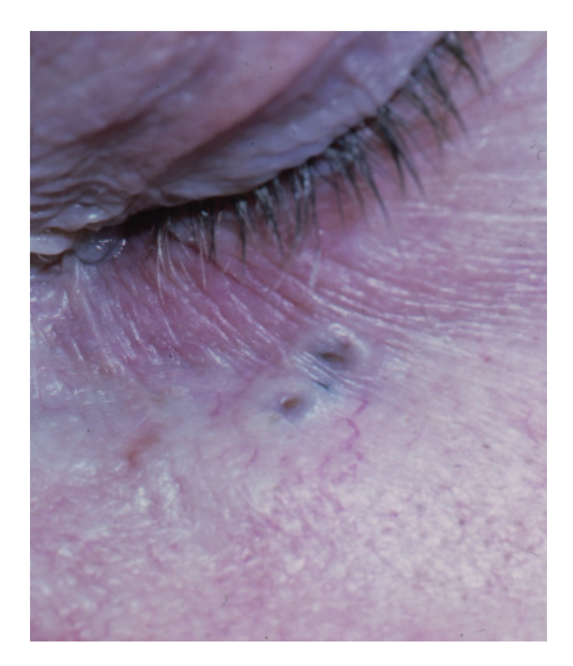


Figure 4.24. BCC.

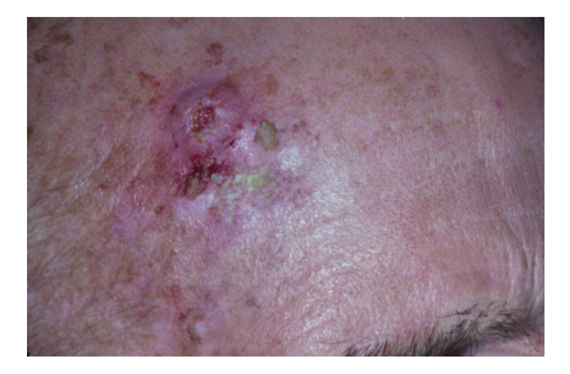


Figure 4.25. BCC.

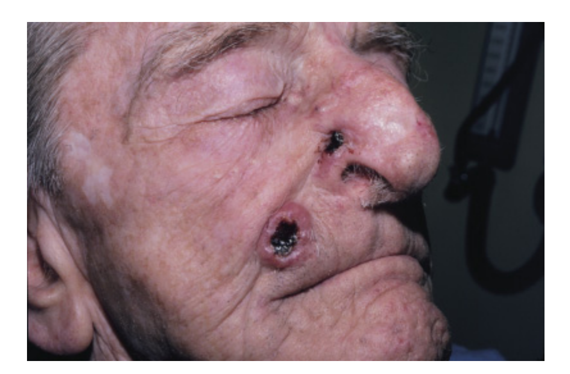


Figure 4.26. BCC.

nevoid BCC syndrome (Gorlin's syndrome) and xeroderma pigmentosum. It has also been reported in children previously treated with ionizing radiation therapy.

Diagnosis
Although BCC can usually be diagnosed by an experienced observer on clinical grounds alone, verification by excisional biopsy is necessary.

Prevention
Patients must be educated about the importance of reducing unprotected exposure to ultraviolet radiation. Sensible measures include sun avoidance, wearing tightly woven clothing, and the use of sunscreens. Patients should be further educated about the early signs and symptoms of cutaneous malignancies and the importance of skin self-examination, with assistance if necessary from hand mirrors or family members in viewing inaccessible areas. Patients with one BCC of the skin are at increased risk of developing new tumors and also are at increased risk for various noncutaneous types of cancer.

Treatment
Surgical excision is the treatment of choice for most primary BCCs. Micrographic surgery is recommended for larger BCCs of

the face and those with more aggressive growth patterns.

Assess Trigeminal Nerve Function

The trigeminal nerve is the sensory nerve of the face, teeth, and mucous membranes. It also provides motor function to the mylohyoid, anterior belly of the digastric, tensor veli palatini, tensor tympani, and muscles of mastication. Branches of the trigeminal nerve convey pain associated with diseases of the teeth and oral soft tissues, eyes, and paranasal sinuses.

Facial and oral trauma may result in paresthesia, an abnormal sensation variably described as tingling, pins and needles, burning, coldness, or a sense of water running over the skin. In addition, the trigeminal nerve may be affected by a disorder known as paroxysmal trigeminal neuralgia.

Sensory Evaluation

With the patient's eyes closed, test for touch (cotton wisp or applicator stick), temperature (warm and cold objects), and pain (pointed object) over the distribution of the ophthalmic, maxillary, and mandibular divisions of the trigeminal nerve. A lack of response (anesthesia), altered response (paresthesia), or increased response (hyperesthesia) may be noted.

Motor Evaluation

Have the patient clench his or her teeth while biting on a tongue blade. The examiner should not be able to pull the tongue blade from the mouth. The left and right sides should be tested separately.

Assess Facial Nerve Function

The facial nerve is the motor nerve of the face (muscles of facial expression), the posterior belly of the digastric, stylohyoid, and stapedius muscles. It also provides secretory motor function to the parotid, submandibular, sublingual, and lacrimal glands. In addition, the facial nerve provides the sense of taste on the anterior two-thirds of the tongue.

Facial trauma and neoplasms in the parotid glands may damage branches of the facial nerve and result in paralysis of the muscles supplied by the affected branches. In addition, transient or permanent seventh nerve damage may result in Bell's palsy characterized by unilateral paralysis of the facial muscles.

Motor Evaluation

Have the patient implement movements of the muscles of facial expression, including smiling, frowning, raising the eyebrows, and closing the eyelids. While the eyes are closed, apply upward pressure to raise the eyebrows. The attempt should be unsuccessful.

Secretory Motor Evaluation

The secretory motor function of the facial nerve is difficult to evaluate, but signs and symptoms of xerostomia, in the absence of other etiologies, warrant consideration.

Evaluate Taste

Have the patient protrude his/her tongue and apply sugar or salt to one side of the anterior two-thirds of the tongue. The patient should experience the taste before retruding the tongue. After a rinse with water, the other side should be tested in a similar fashion.

Examine the Ears and Temporomandibular Joints

Hard nodules on the helix may represent sodium biurate deposits suggestive of gout. Eroded areas on the ears may be associated with basal cell carcinoma or squamous cell carcinoma, both of which may cause distortion or destruction of the auricle. Develop-

mental deformities are rare, but when encountered, they may be associated with malformations of the genitourinary tract with head and neck/facial manifestations (i.e., Treacher-Collins syndrome).

A hematoma along the mastoid process is indicative of a temporal bone fracture. A bloody but watery discharge from the ears and nose is likely to represent cerebrospinal fluid that has escaped through a meningeal tear caused by a skull fracture. Palpation of the temporomandibular joint as the patient attempts to move the mandible may reveal a dislocation. Limitation of movement may indicate other pathological conditions.

Assess Acoustic Nerve Function

The acoustic nerve provides the special sense of hearing (auditory) and the special sense of balance (vestibular). Vestibular lesions cause vertigo (dizziness) and unbalance. Deafness may either be due to faulty conduction or nerve damage. Conduction deafness is usually a sequel to inflammatory middle ear disease. It may also be secondary to obstruction of the Eustachian tube, otosclerosis, or Paget's disease. Nerve deafness may be congenital, drug-induced, or caused by fractures of the temporal bone. Ménière's disease, or a gross distention of the labyrinth by fluid, is characterized by deafness, tinnitus (ringing in the ears), and vertigo. It occurs most often in middle-aged women.

Evaluate Air Conduction

A simple yet effective way to quickly assess air conduction is to hold a ticking watch about 8 inches from the patient's ear (in a quiet room). Repeat on the opposite side. The perception should be the same in each ear.

Evaluate Bone Conduction

Hold a ringing tuning fork against the top of the patient's head. It should be heard equally in both ears. Alternatively, the struck tuning fork should be held against the mastoid process until the patient can no longer hear it. It should then be held next to the ear and the patient should hear it again (air conduction is about three times more efficient than bone conduction). Repeat on the opposite side.

Examine the Nose

Respiratory abnormalities may be associated with dilation of the nostrils upon inspiration and contraction of the nostrils upon expiration. A deviated septum, allergies, or nasal polyps may cause chronic nasal obstruction. Basal cell carcinoma is the most common malignancy affecting the skin of the nose. A "butterfly" lesion on the skin of the nose extending out over the cheeks is characteristic of **lupus erythematosus**.

Note a deviated or depressed nasal bone or cartilage, which along with extraocular movements may indicate a fractured or displaced facial bone. A bloody but watery discharge from the nose and ears is likely to reflect cerebrospinal fluid that has escaped through a meningeal tear caused by a skull fracture.

Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a B-cell mediated autoimmune disease. It is characterized by the presence of circulating immune complexes and activation of the complement system, which can affect the joints, skin, lungs, heart, and kidneys. In North America and northern Europe, SLE occurs in about 40 persons per 100,000. Women are nine times more likely to be affected by SLE than men, and blacks are more commonly affected than Caucasians. The etiology of SLE remains unclear, although a genetic predisposition is likely. Other etiologic risk factors include environmental exposures (e.g., infectious, dietary, or toxic), ultraviolet light, and exposure to certain drugs (e.g., procainamide).

The autoantibodies in SLE are predominantly of the immunoglobulin G (IgG) type. These autoantibodies can react with a variety of cellular and extracellular constituents, including DNA and other nucleic acids, nucleoproteins, cytoplasm components, cell surface antigens, and matrix components, and initiate an inflammatory response that often leads to cell death and failure of affected organs.



Figure 4.27. Acute cutaneous LE.

Clinical Features

SLE can strike suddenly and severely but is more likely to cause mild, chronic illness for a long period of time before being diagnosed. Skin rashes in lupus come in all shapes and sizes and can be divided into three categories. Acute cutaneous LE is characterized by the classic photosensitive "butterfly" rash of SLE (Figure 4.27). Subacute cutaneous LE (SCLE) is characterized by nonscarring, photosensitive annular (red rings) or papulosquamous (red scaly) lesions. About 10% of these cases progress to full-blown SLE. The most common form, chronic cutaneous LE, also termed discoid LE (DLE), is characterized by scarring usually localized to the scalp, face, and ears (Figures 4.28a–4.28c).

Other manifestations include flu-like symptoms with fever, tiredness, headaches, muscle and joint pain, transient hair loss, arthritis, vasculitis, cardiac and renal involvement, blood disorders (anemia, leukopenia, and thrombocytopenia), and emotional disturbances.

Oral lesions may occur and are generally described as annular leukoplakic areas or erythematous erosions. These lesions may present diffusely throughout the oral cavity, affecting the buccal mucosa, tongue, palate, and lips, or may present as a prominent marginal gingivitis.

Diagnosis

Due to its widely variable presentation, the diagnosis of SLE may be difficult. It should be suspected in any patient who has features affecting two or more organ systems. The diagnosis of SLE is based on correlating the presenting signs and symptoms with appropriate laboratory testing. The presence of anti-double-stranded DNA antibodies is a specific diagnostic test, and is also used to monitor lupus activity.

Treatment

There is currently no cure for SLE. Therapy is aimed at relieving symptoms with the hope of controlling the disease so that patients can lead a normal life. Patients should avoid sunlight and wear a broad-spectrum sunscreen, when necessary. Medications prescribed to manage patients with SLE may include NSAIDs, antimalarials (hydroxychloroquine), anticoagulants, corticosteroids,



Figures 4.28a–c. Chronic cutaneous LE.

and immunosuppressants (azathioprine, cyclophosphamide).

Assess Olfactory Nerve Function

The olfactory nerve provides the special sense of smell. The sense of smell may be impaired with age, upper respiratory tract infections, allergies, smoking, and medications. Loss of the sense of smell (anosmia) may also occur as a result of a fracture of the base of the skull or pressure on the olfactory nerve by a tumor.

Evaluate Smell

Occlude one nostril and with the patient's eyes closed ask him or her to identify familiar substances (coffee, tobacco) by smell. Repeat on the opposite side.

Examine the Eyes

Increased monocular blinking often follows physical irritation of the eye, while bilateral blinking is often associated with the wearing of contact lenses. Decreased monocular blinking is seen in patients with Bell's palsy or a facial nerve deficit. Conjunctivitis occurs in response to physical irritants, specific allergens, or infections. Focal lid edema is often due to contact allergy. Xanthelasmic plaques, most frequently seen on the lower lids, suggest dyslipidemia. Ptosis, unilateral or bilateral drooping of the eyelids, may be an early manifestation of **myasthenia gravis**. Blue sclera may be associated with osteogenesis imperfecta or **dentinogenesis imperfecta**. A yellow sclera suggests icterus. Exophthalmia is an abnormal protrusion of the eyes. When only one eye is affected, the likely

cause is a tumor. When both eyes are affected, the most likely cause is **hyperthyroidism**. Compare the orbital rims and zygomatic arches for symmetry and note extraocular movements. Abnormalities may indicate a fractured or displaced facial bone.

Myasthenia Gravis

Myasthenia gravis (MG) is a rare but potentially debilitating autoimmune disease of the neuromuscular junction characterized by an impaired transmission of the neuron-to-muscle impulse. In MG, the acetylcholine receptors at the postsynaptic nerve membrane are blocked by acetylcholine receptor antibodies. The estimated prevalence of MG varies from 50 to 150 cases per million. Approximately 80% of patients with MG have hyperplasia of the thymus and 15% have thymomas. MG may also be associated with other disorders such as rheumatoid arthritis, pernicious anemia, or SLE. Certain medications, including aminoglycosides, ciprofloxacin, lithium, phenytoin, procainamide, quinidine sulfate, and beta-adrenergic receptor-blocking drugs (including eye drops) may exacerbate MG.

Clinical Features

MG is characterized by progressive fatigue with exercise. The signs and symptoms may range from isolated ptosis (Figure 4.29a), diplopia, or mild proximal muscle weakness to severe generalized weakness. More generalized disease usually affects muscles that control neck movement, facial movement, mastication, and the tongue (Figures 4.29b–4.29d). A smile resembles a snarl, chewing is impaired, and swallowing or speaking becomes difficult. Involvement of the vocal cords gives speech a nasal quality. Disease involving the muscles in the limbs, neck, shoulders, hands, diaphragm, and abdomen may lead to difficulty walking, sitting up, and breathing.

Diagnosis

The diagnosis of MG should be considered in all patients with ptosis or ocular motor weakness without pupillary involvement or in older patients presenting with any bulbar or skeletal muscle weakness and a history of recurrent falls. Testing for acetylcholine-receptor antibodies is specific and detectable in 80–95% of patients with generalized MG and in 34–56% of patients with isolated ocular involvement. MG should be differentiated from a stroke and other motor neuron diseases.

Treatment

Therapy for MG is directed at reducing the degradation of acetylcholine with cholinesterase inhibitors, thereby increasing its duration of activity in the neuromuscular junction. Drugs implicated in possibly exacerbating MG should be avoided. Four methods of treatment are currently in use: (1) enhancement of neuromuscular transmission with anticholinesterase agents; (2) surgical thymectomy; (3) immunosuppression (corticosteroids, azathioprine, and cyclosporine); and (4) short-term immunotherapy, including plasma exchange and intravenous immune globulin.



Figures 4.29a–d. Myasthenia gravis.

Dentinogenesis Imperfecta

Dentinogenesis imperfecta (DI) is a genetic disorder of tooth development and affects an estimated 1 in 6,000 to 8,000 individuals. Three forms of DI are recognized. DI Type I occurs in association with osteogenesis imperfecta, a disease caused by a type 1 collagen defect. DI Types II and III exist as isolated dentition-related diseases attributed to mutations affecting the dentin sialophosphoprotein (DSPP) gene. DSPP encodes two tooth matrix proteins (dentin sialoprotein [DSP] and dentin phosphoprotein [DPP]) that are associated with tooth mineralization. Recent studies have shown that DI Types II and III are associated with DSPP mutations affecting DSP and DPP production, respectively.

Clinical Features

All three forms of DI manifest an amber-brown to blue-gray hue to the teeth with an



Figures 4.30a–e. Dentinogenesis imperfecta.

opalescent sheen, cracking/loss of enamel, and attrition (Figures 4.30a–4.30c). Radiographically, the teeth in DI Type I exhibit bulbous crowns, cervical constrictions, short roots, obliteration of pulp chambers, and periradicular radiolucencies (Figures 4.30d and 4.30e). In Type III DI the pulp is normal or nonmineralized (shell-teeth).

Diagnosis

The dentist will often be the first to recognize the characteristic clinical presentation of DI. The presence of blue sclera and/or a history of increased bone fragility, in association with DI, should raise the suspicion of osteogenesis imperfecta, and an appropriate

medical referral should be initiated to rule it out.

Treatment

DI Types II and III pose no threat to longevity but may present significant challenges in terms of restoring esthetics and function.

Hyperthyroidism

Hyperthyroidism is a condition caused by overactivity of the thyroid gland. In contrast, thyrotoxicosis refers to any state of thyroid hormone excess, including ingestion of exogenous thyroid hormone and thyroiditis (inflammatory changes within the thyroid gland). Hyperthyroidism is common, affecting approximately 4.5 million Americans, and there is a clear predilection for women (5–10 : 1). The most frequent cause of hyperthyroidism is Graves' disease, an autoimmune disease caused by the production of autoantibodies against the TSH receptor. Other causes of hyperthyroidism include toxic nodular goiter and thyroiditis.

Clinical Features

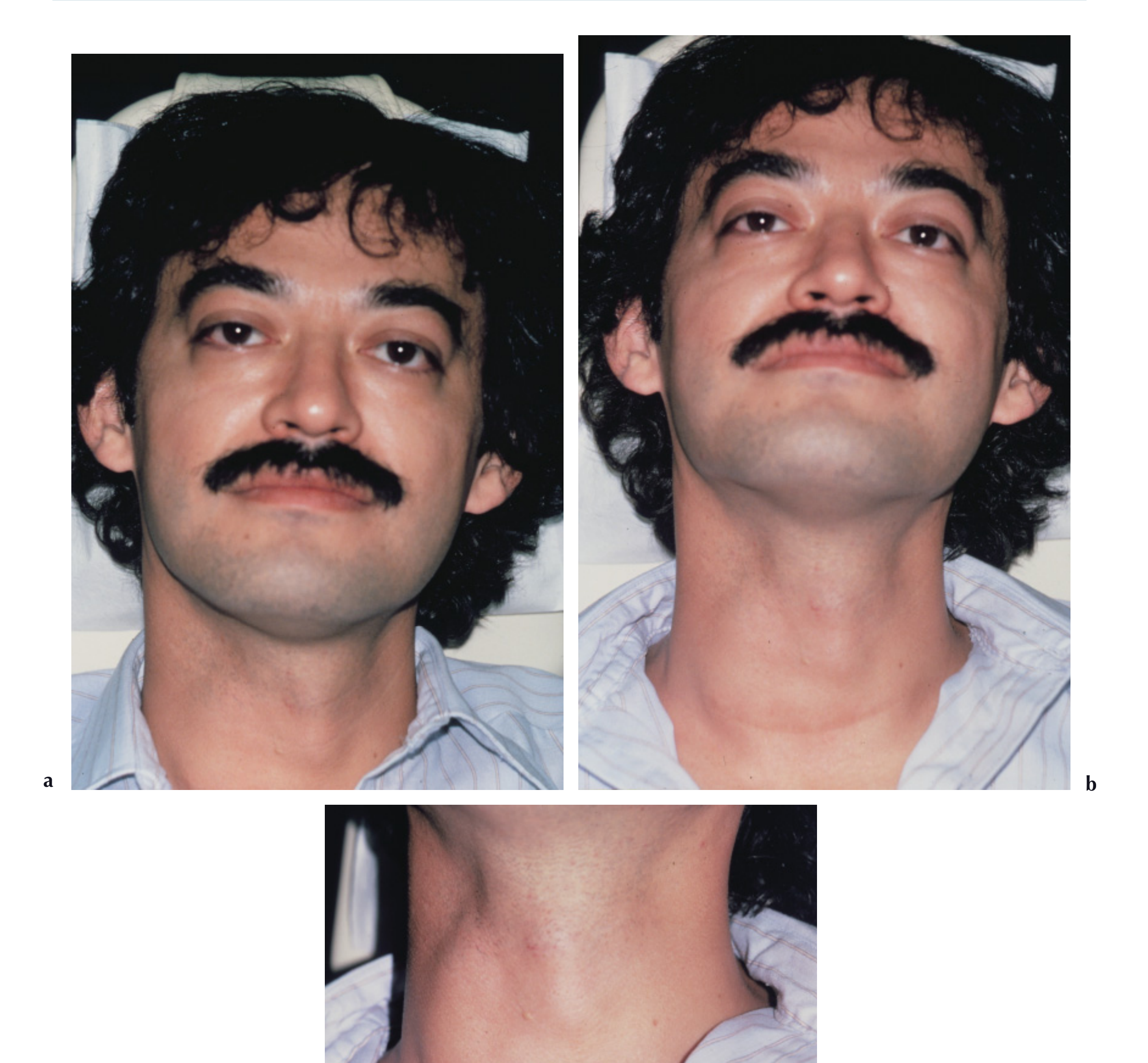
Common signs and symptoms of hyperthyroidism include nervousness, increased sweating, heat intolerance, palpitations, dyspnea, fatigue, weight loss, exophthalmos, and unilateral or bilateral enlargement of the thyroid gland (Figures 4.31a–4.31c). Unfortunately, hyperthyroidism may develop insidiously over time, and the classic signs and symptoms may be easily overlooked.

Diagnosis

A diagnosis of hyperthyroidism is suggested by the presence of the classic signs and symptoms, which occur in 50% of patients. Appropriate laboratory testing is essential and is characterized by low levels of TSH and high levels of free thyroxin (T_4).

Treatment

Treatment of hyperthyroidism is aimed at attaining a state of euthyroidism by decreasing circulating thyroid hormone levels. Current options include antithyroid medications, radioactive iodine (^{131}I), or subtotal thyroidectomy. The type of treatment rendered is determined by the underlying cause, the age of the patient, the size of the goiter, and the presence of coexisting morbidities. A potential side effect of any therapy, but especially ^{131}I, is hypothyroidism. Hypothyroid patients will require supplemental thyroid hormone replacement therapy to obtain a euthyroid state.



Figures 4.31a–c. Hyperthyroid.

Assess Optic Nerve Function

The optic nerve provides the special sense of vision. Changes in visual acuity may be associated with diseases such as hypertension, renal disease, blood dyscrasias, glaucoma, and diabetes mellitus. Visual field impairment may be caused by optic nerve impingement associated with a pituitary tumor or Paget's disease. Acute oculomotor palsy and/or visual disturbances may develop as a complication of the injection of local anesthesia into the infraorbital region. Oculomotor palsy manifests as diplopia, strabismus, eye deviation, impaired ocular movement, and ptosis. Visual disturbances include blurred vision, central vision loss, and altered color vision.

If an ocular complication develops in the oral healthcare setting, the patient should be reassured that this complication is usually transient, and the affected eye should be covered with a gauze dressing to protect it until the anesthesia wears off. Because monocular vision limits distance perception, an adult should escort the patient home. If the complication lasts for more than 6 hours, a referral to an ophthalmologist is mandatory.

Evaluate Visual Acuity

Test visual acuity by asking the patient to read a passage from a newspaper. This test is performed with both eyes uncovered and then with each eye covered in turn.

Evaluate Visual Field

The patient is instructed to stare at the examiner's nose. The examiner moves his or her index finger into the patient's visual field from the extreme periphery to the center starting at each quadrant around the circle. The patient is instructed to indicate initial sighting of the finger as well as any loss of visual continuity. Normal visual fields are 60° to the medial (nasal) side, 100° to the lateral (temporal) side, and 130° vertically.

Assess Oculomotor, Trochlear, and Abducens Nerve Function

The oculomotor nerve controls the papillary sphincters and ciliary muscle and provides motor function to the superior, inferior and medial rectus, inferior oblique, and levator palpebrae muscles. The trochlear nerve provides motor function to the superior oblique muscle. The abducens nerve provides motor function to the lateral rectus muscle.

Evaluate the Pupils

Using a point source light in a somewhat darkened room, test each eye to ensure that the pupils are equal, round, and reactive to light, and to confirm accommodation (PERLA). The pupils may be constricted, as in a drug overdose; unequal, as in a stroke; or dilated, as in shock and unconsciousness. Coloboma is a congenital ocular malformation presenting as an out-of-round pupil. Less commonly, it may result from injury or surgery to the eye.

Evaluate Ocular Movement

The patient should be instructed to follow the examiner's index finger vertically, horizontally, and diagonally. If the oculomotor nerve is damaged, the patient will exhibit ptosis, pupillary dilation, and an inability to move the eye up, down, and medially. If the trochlear nerve is damaged, the patient will be unable to rotate the eye diagonally, downward, and laterally. If the abducens nerve is damaged, the eye will not abduct. A neurological deficit in any of these nerves will manifest as diplopia. Strabismus, or deviation of the eyes in relation to one another, may be indicative of muscle paresis. Ptosis may be an early sign of myasthenia gravis. Nystagmus, or oscillation of the eyes, is a pathologic sign of a neurological disorder. All these findings warrants referral to an ophthalmologist.

Examine the Hair

Male-pattern baldness is inevitable in the presence of a genetic predisposition. Distinct patches of baldness may result from chemical or radiation burns and are common in ringworm infestations of the scalp. Sudden, patchy, idiopathic loss of hair (**alopecia areata**), may be related to stress. Sparse hair may be noted in patients with pituitary insufficiency and **ectodermal dysplasia**. Scant, coarse, dry, and lusterless hair should suggest hypothyroidism.

Alopecia Areata

Alopecia areata (AA) is a remitting, recurring loss of hair with an unpredictable prognosis. There is growing evidence to suggest that AA is a tissue-restricted autoimmune disease, mediated by a T-lymphocyte response to a follicular autoantigen. A family history of AA occurs in 10–42% of cases and in identical twins, the concordance rate of AA is as high as 55%. Other conditions associated with AA are allergic diseases (atopy, asthma, and eczema). Overall, an estimated 1.7% of the population has AA, there is no sex predilection, and it is most common in children and young adults.

Clinical Features

AA usually presents as small, round patches of hair loss on the scalp (Figures 4.32a–4.32c). In about one-fifth of patients with AA, the condition evolves into complete scalp hair loss (alopecia totalis), and in some cases the loss of all body hair may occur (alopecia universalis). Other associated clinical changes may include nail dystrophy, which usually presents as fine pitting or ridges on the surface of the nail. The course of AA is unpredictable, with periods of remission and exacerbation. The rate and pattern of hair loss is variable. New hair growth may show loss of pigment.

Diagnosis

The diagnosis of AA is usually made on the basis of clinical history and physical examination. A biopsy may be helpful to rule out other conditions. A differential diagnosis should include infection, traction (e.g., tight braids, pony tails), stress, hormonal



Figures 4.32a–c. Alopecia areata.

disturbances, and psychiatric illness (trichotillomania).

Treatment

About half of AA patients experience spontaneous return of hair growth without therapy. Treatment for alopecia areata includes steroids (topical, intralesional, and systemic), minoxidil, anthralin, and immunotherapy. However, in some cases, AA progressively worsens in spite of therapy.

Ectodermal Dysplasia

Ectodermal dysplasia (ED) represents a rare group of inherited disorders characterized by aplasia or dysplasia of ectodermallly derived tissues. Hair, nails, teeth, and skin are often affected. The mode of inheritance varies among the different types of ED. Ectodermal dysplasia may be inherited as an X-linked recessive trait (hypohidrotic ED), in which case the gene is carried by the female and manifested in the male. However, there are reports of multiple siblings being affected and of females suffering from this condition. It is likely that most of these cases are examples of the autosomal-recessive form (hidrotic ED) of the disease. The prevalence in the general population has been assessed as between 1 in 10,000 and 1 in 100,000 live male births.

Clinical Features

The X-linked hypohidrotic form (Christ-Siemens-Touraine syndrome) of ED is the most common and is characterized by the classic triad of hypohidrosis (abnormal or missing sweat glands), hypotrichosis (abnormal hair), and hypodontia. The facial manifestations of ED include a prominent forehead, sparse and fine blonde hair, depressed nasal bridge, high/broad cheekbones, pointed chin, and protuberant lips (Figures 4.33a–4.33c,). The less common hidrotic form (Clouston syndrome) does not typically involve sweat glands but does affect teeth, hair, and nails.

Other signs and symptoms may include rhinitis, pharyngitis, asthma, epistaxis, and hearing loss (resulting from an accumulation of wax in the auditory canal). Xerostomia is an uncommon complaint because complete absence of the salivary glands is rare. Dental findings in ED may range from anodontia, to, more commonly, hypodontia of the primary or permanent teeth (Figures 4.33d and 4.33e). Teeth that are present often have conical crowns. Other defects include high palatal arch or even a cleft palate, reduced vertical dimension because of the absence of teeth, and enamel hypoplasia.

Diagnosis

The diagnosis of ED is typically straightforward and established in early childhood. At least two clinical features of the components (hypodontia, hypotrichosis, and hypohidrosis) must be present in order to establish the diagnosis of ED. Conditions to consider in the differential include sporadic oligodontia, exposure to head and neck irradiation during childhood, chondroectodermal dysplasia, and cleidocranial dysplasia.

Treatment

The dental management of ED is both complex and challenging. The major goal is to provide the patient with optimal esthetics and function to foster normal physical and emotional growth. Implant-supported prostheses may offer the best opportunity for successful rehabilitation. The major advantage of implant-supported prostheses is increased retention and stability of the prostheses, leading to improvement in function and esthetics. It has been suggested that dental and skeletal maturity, not chronological age, should be the determining factor if implants are to be considered. When feasible, fixed restorations allow ED patients to avoid social problems that are associated with partial or full dentures, particularly in younger patients.



Figures 4.33a–e. Ectodermal dysplasia.

Examine the Neck

Inspect the neck for asymmetry, unusual pulsations, or limitations of motion. By simply extending and turning the neck, tension of the sternocleidomastoid muscle brings into view the boundaries of the anterior and posterior triangles. The carotid arteries often visibly pulsate if there is aortic insufficiency, or they may throb in patients who have severe anemia, atherosclerosis, arteriosclerosis, or hyperthyroidism.

Upon palpation, enlargement of the thyroid gland or abnormalities of the vascular structures may become apparent. Normally, the thyroid gland is a firm, smooth, midline mass that moves upward with swallowing. A **thyroglossal duct cyst** typically presents as a fluctuant midline swelling and represents residua of the thyroglossal tract following descent of the thyroid gland from the base of the tongue. A **branchial cleft cyst** appears in the upper lateral neck just anterior to the sternocleidomastoid muscle. Anatomically, the inferior lobe of the submandibular gland lies inferior to the posterior attachment of the mylohyoid muscle. Inflammation or a **sialolith** associated with the inferior lobe of the submandibular gland may become apparent in the neck.

Thyroglossal Duct Cyst

The thyroglossal duct cyst (TDC) is the most common developmental nonodontogenic cyst to occur in the neck. It accounts for approximately 40% of cervical malformations in children and has a peak incidence during the first 3 decades of life. Men and women are equally affected. It usually presents as an asymptomatic midline mass in the anterior neck at the level of the thyroid gland. However, it may arise anywhere along the thyroglossal tract, from the base of the tongue (lingual thyroid) to the anterior neck.



Figure 4.34. Thyroglossal duct cyst.

Clinical Features

The TDC usually presents as a painless, well-defined, smooth, asymptomatic midline swelling in the neck (Figure 4.34). About 25% of cases are present at birth and another 30% are diagnosed by the age of 10 years. They may be found anywhere in the midline from the submental region to the suprasternal notch, but most are found near the hyoid bone. Only 1% of TDCs are lateral to the midline. With swallowing or protrusion of the tongue, a TDC classically rises in the neck as a result of the cyst being anchored to the hyoid bone and the muscles of the tongue. If a TDC is secondarily infected, drainage to the overlying skin through a sinus tract may occur. Approximately 1% of TDCs undergo neoplastic change, usually to form a papillary adenocarcinoma.

Diagnosis

The presence of a midline mass that undergoes upward movement upon swallowing is highly suggestive of a TDC. The diagnosis is usually confirmed with a fine needle aspiration biopsy. Radiographic studies are useful to delineate extent of the lesion and the

presence of ectopic functioning thyroid tissue. Other lesions to consider in a differential diagnosis include a dermoid cyst, ectopic thyroid tissue, lipoma, sebaceous cyst, lymph node, branchial cleft cyst, and autoimmune thyroiditis.

Treatment

Treatment of a TDC is complete surgical excision. It is often recommended that the central portion of the hyoid bone be removed in an effort to eliminate any residual thyroglossal tract epithelium and avoid recurrence. Indications for excision may include cosmetic appearance, recurrent infections, sinus tract formation, and the risk of malignant transformation. Routine follow-up care is recommended to monitor for recurrence.

Branchial Cleft Cyst

Branchial cleft cyst is a congenital developmental defect that arises from the primitive branchial apparatus (usually the second branchial arch). It is believed to reflect incomplete closure of clefts and pouches or a failure of obliteration of the cervical sinus. It is an epithelial lined cavity, which may be associated with a draining sinus tract.

Clinical Features

The branchial cleft cyst typically presents as a soft, round mass located along the anterior border of the sternocleidomastoid muscle (Figure 4.35a and 4.35b). It can increase in size during an upper respiratory tract infection. It may also appear in the submandibular area, adjacent to the parotid gland, or around the sternocleidomastoid muscle. A branchial cleft cyst usually becomes clinically apparent in late childhood or early adulthood. Computed tomography is useful to delineate the extent of the lesion.

Diagnosis

The typical lateral presentation of a branchial cleft cyst is highly suggestive of the correct diagnosis. However, a definitive diagnosis is usually obtained after histological examination of the excised tissue. Differential diagnosis should include cervical lymphadenitis, dermoid cyst, retropharyngeal abscess, skin inclusion cyst, lymphangioma, and primary or metastatic parotid tumor.

a



b



Figures 4.35a and b. Branchial cleft cyst.

Treatment

Complete excision is the treatment of choice. The overall recurrence rate is about 3–5%, with a slightly increased risk of recurrence when a sinus tract is present.

Sialolith

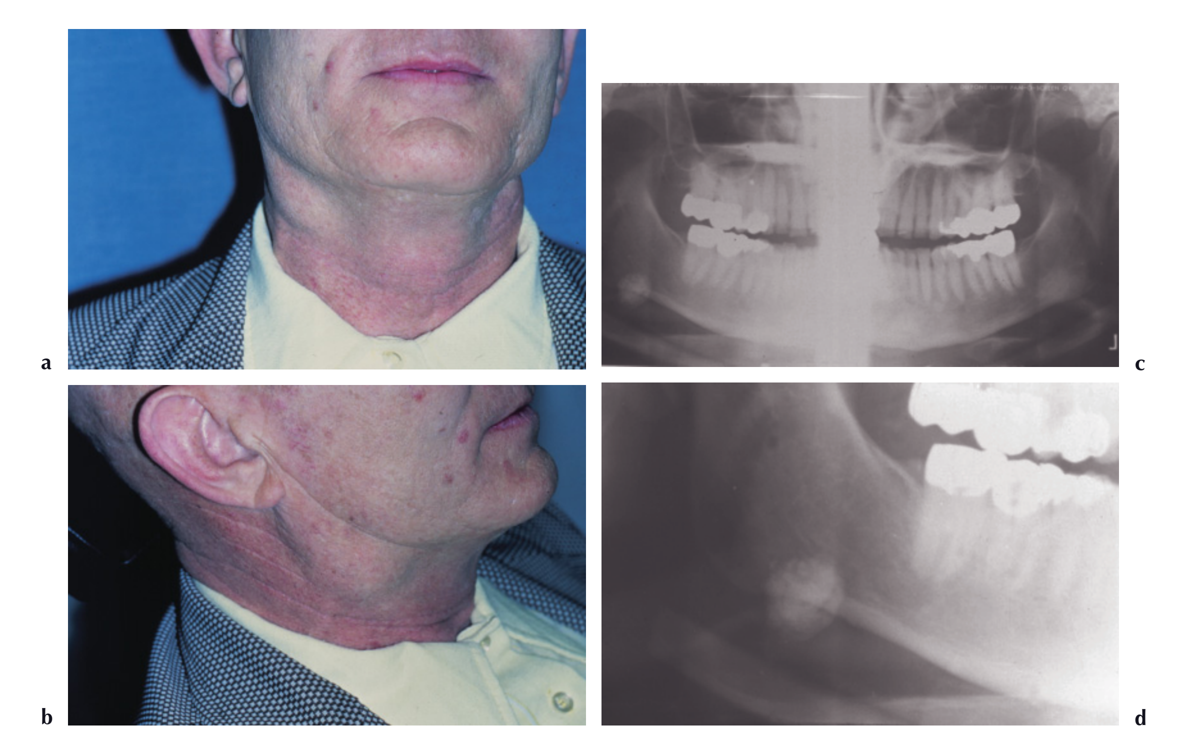
Sialoliths are a major cause of salivary gland dysfunction. They are spherical, calcareous deposits within the ductal system or parenchyma of major or minor salivary glands. It is believed that they are derived from intracellular microcalculi that when excreted into the ductal system provide a platform for further calcification. Other theories suggest that "mucous plugs" within the ductal system or bacteria from the oral cavity that migrated into the ductal system become the nidus for further calcification. Multiple sialoliths occur in approximately 25% of patients.

Clinical Features

The most characteristic complaint is a painful swelling within the affected gland just before, during, and immediately after meals (Figures 4.36a and 4.36b). In a minority of cases, symptoms are absent. A sialolith is detected by either bidigital palpation of Wharton's or Stensen's ducts, or by radiographic examination (Figures 4.36c and 4.36d). If the duct (usually Wharton's duct) becomes completely occluded, retrograde movement of oral bacteria in the duct may result in acute sialadenitis. Untreated sialadenitis may lead to fibrosis and atrophy of the affected gland.

Diagnosis

The most important considerations in the diagnosis of obstructive salivary gland disease are patient history and clinical examination. Palpation of the affected gland may reveal reduced flow from the affected duct. The



Figures 4.36a–d. Sialolith.

presence of cloudy or purulent flow usually indicates the presence of secondary infection. Radiographs may be useful to demonstrate a sialolith. However, while submandibular sialoliths are usually radiopaque, parotid blockages are usually caused by mucous plugs and thus are not visible on radiographs.

Treatment

Management of a sialolith within the submandibular gland depends on the duration of the symptoms, the size of the stone, and the location of the stone. Small sialoliths may be successfully excreted or removed while preserving the gland and associated duct. Larger sialoliths necessitate surgical removal of the affected gland. Newer, less invasive techniques include interventional sialendoscopy and ultrasound-guided piezoelectric extracorporeal lithotripsy.

Assess Spinal Accessory Nerve Function

The spinal accessory nerve provides motor function to the sternocleidomastoid and trapezius muscles. Injury to the nerve may manifest as paralysis of these muscles.

Evaluate Motor Function

Ask the patient to rotate the head to one side and palpate the sternocleidomastoid muscle while applying counter-rotational pressure to the head. Repeat on the opposite side. In a similar manner, the size and strength of the trapezius muscles may be palpated as the patient shrugs the shoulders against resistance provided by the examiner. The muscles should not yield to the examiner's downward pressure.

Examine the Lymph Nodes

After a general evaluation of the head and neck has been completed, but before the oral cavity is examined, the various lymph nodes in the head and neck region should be palpated (Figure 4.37) Lymphadenopathy associated with intraoral pathoses primarily involves the submandibular, submental, and anterior cervical nodes.

Lymphadenopathy is an indication of an abnormality that must be identified (Table 4.3). Pathological changes in cervical lymph nodes may represent an inflammatory, degenerative, or neoplastic process. Neoplastic involvement of cervical nodes may occur as a primary process such as **lymphoma**. Secondary involvement of lymph nodes in the neck may be seen in association with intraoral **squamous cell carcinoma**.

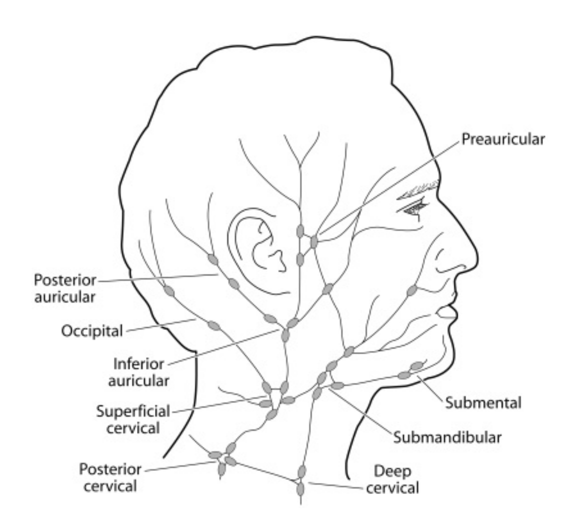


Figure 4.37. Lymph nodes.

Table 4.3. General conditions suggested by palpable nodes.

Condition	Nodal characteristics
Acute inflammatory disease	Smooth, firm, movable, and tender node
Past or present chronic disease with fibrosis	Discrete, firm, movable, and nontender node
Neoplasm	Matted, nontender, usually firm and fixed node
Systemic disease	Variably palpable, usually nontender node

Lymphoma

Lymphoma is a neoplasm of lymphoid cells found in the lymph nodes, spleen, liver, and bone marrow. It is defined by uncontrolled growth of a specific lymphocyte lineage. While there are several specific types of lymphoma, all are classified as either being a Hodgkin's or a non-Hodgkin's lymphoma. Over 71,000 cases of lymphoma are diagnosed in the United States each year, and over 88% of these cases are of the non-Hodgkin's type. Both types of lymphoma demonstrate a slight male predilection. Like most other malignancies, the etiology of lymphoma is only partially understood, and numerous genetic and environmental factors likely contribute to the disease. As many cases of lymphoma initially present in the head and neck, the dental practitioner is often in a unique position to be the first to suspect its presence and initiate a medical referral.

Clinical Features

The signs and symptoms of lymphoma are often indolent and include swollen, painless lymph nodes in the neck (Figure 4.38), armpits, or groin; unexplained weight loss; fever; soaking night sweats; chronic cough and breathing difficulties; persistent fatigue; pruritis; and fullness or pain in the abdomen. Oral involvement is rare, but if present, typically manifests as a progressive swelling or mass that may undergo ulceration (Figure 4.39).



Figure 4.38. Hodgkin's lymphoma.

Diagnosis

Lymphoma should be considered in any patient with the signs and symptoms listed above. A biopsy of a suspicious lymph node is required to establish a definitive diagnosis. This may be accomplished by performing either a simple excisional biopsy or a fine needle aspiration. Once the diagnosis is obtained, an extensive medical workup is undertaken to determine the stage (spread) of the lymphoma.

Treatment

The treatment and prognosis of a patient with lymphoma is dependent on both the type and the stage of the disease. Potential therapeutic measures include surgery, chemotherapy, radiation therapy, immunotherapy, and bone marrow or stem cell transplantation. Overall, the survival rate of lymphoma has gradually improved over the past few decades and now exceeds 60%.



Figure 4.39. Hodgkin's lymphoma.

Squamous Cell Carcinoma

Squamous cell carcinoma (SCC) is an invasive tumor with metastatic potential. It is derived from the thin, flat squamous cells that are found on the skin surface; and the mucosal lining of hollow organs such as the respiratory and digestive tracts, including the oral cavity.

Over 90% of oral and pharyngeal cancers are SCCs. Tobacco, particularly cigarette smoking, is the major risk factor associated with the development of oral SCC. Additionally, a dose-risk relationship between tobacco and/or alcohol use and the development of oral cancer has been noted. Other studies implicate human papillomaviruses (HPVs), most frequently HPV-16 and HPV-18, in the pathogenesis of some oral SCC. Additional risk factors such as living in rural areas, socioeconomic status, age, gender, and humoral and cellular immune mechanisms may play less well understood roles. Chronic periodontal disease, poor oral hygiene, ill-fitting dentures, sharp teeth, electrogalvanism, and edentulism have been suggested as cofactors.

Oral cancer consistently ranks as one of the top ten cancers worldwide, with broad differences in geographic distribution. In the United States, the number of new cancer cases for 2007 reached 1,444,920. Of these newly diagnosed cases, approximately 34,360 were malignancies of the oral cavity and pharynx. While this represents less than 3% of all malignant neoplasms diagnosed annually in the United States, in developing countries the incidence is much higher. Oral cancer remains predominantly a disease of males. However, the male to female ratio has steadily shifted from about 6 : 1 in 1950 to about 2 : 1 in 1997. The changing ratio is likely the result of the increase in smoking among women in the past three decades. Most cases of oral cancer in the United States are diagnosed in the sixth and seventh decades of life, with the highest prevalence noted in patients over 65 years of age. A recent study in the United States reported an alarming increase in the incidence of oral cancer, particularly tongue cancer, in young white males under the age of 40.

Clinical Features

SCC may appear as a flat leukoplakia, erythroplakia, or speckled leukoplakia, which may evolve into a persistent nodule, tumor, or indurated ulcer (Figures 4.40a and 4.40b). Symptoms are uncommon in earlier stages of the disease but become more frequent with local invasion. In particular, paresthesia and anesthesia in the absence of a history of trauma are highly suggestive of invasive SCC. Metastatic dissemination occurs through the submandibular, cervical (Figure 4.40c), and jugular lymphatic pathways, and distant metastases most commonly spread to the lungs. The majority of oral SCCs originate from nonkeratinized mucosa. The three most common sites of involvement are the tongue (30%), lip (17%), and floor of the mouth (14%). Recently, a trend toward an increased number of lesions arising on both the dentate and edentulous gingiva has been reported.

Diagnosis

The responsibility to identify any suspicious oral lesion clearly falls under the purview of a dental professional. Any unusual oral lesion lasting more than 2 weeks must be regarded with suspicion, and a biopsy should be performed to rule out malignancy. To safeguard and advance the welfare of the patient, a clinician who is uncomfortable performing a biopsy has the obligation to refer the patient to a respected peer or specialist with special skills, knowledge, and experience in managing oral cancer.

Several adjunctive diagnostic modalities (toluidine blue vital staining, autofluorescence, oral brush biopsy, chemiluminescence, computed tomography, and magnetic resonance imaging) are currently marketed to aid the clinician in increasing the diagnostic yield. However, no modality, not even the current recommendation to perform a routine oral cancer screening examination, has been



Figures 4.40a–c. SCC.

conclusively validated as a cost-effective screening method to diagnose oral cancer.

Treatment

Once the diagnosis of SCC has been established, the patient will need to undergo a thorough medical evaluation to stage the neoplasm. Staging is an essential component in the management scheme and helps to determine prognosis and guide therapy. Typically, head and neck cancer is treated by one or more of a combination of the three principal therapeutic modalities: surgery, radiotherapy, and chemotherapy. The use of one treatment over another depends on the size, location, and stage of the primary tumor; the patient's ability to tolerate treatment; and the patient's desires.

Surgical excision is the preferred modality for most well-defined and accessible solid tumors. However, it has its limitations for inaccessible or more advanced tumors demonstrating lymph node involvement and/or metastasis. For such cases, radiotherapy may be either an effective alternative to surgery or a valuable adjunct to surgery and/or chemotherapy in the loco-regional control of SCC. While the benefit of neoadjuvant (induction) chemotherapy has been recently scrutinized, several studies have shown that concomitant chemo-radiotherapy improves both loco-regional control and survival.

Conclusion

Physical signs are produced by physical causes. Since physical problems are the determinants of physical signs, these signs and symptoms must be recognized before the physical problems can be diagnosed and treated.

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5 Examination of the Oral Cavity

Dentists should have a special interest in the physical examination of the oral cavity since the mouth is the anatomical area of the body for which they are the ultimate authority. Therefore, the organization of this section is more detailed and provides greater emphasis on possible findings and interpretation of data. Basic instrumentation for the oral examination includes a good light source, a mouth mirror, an explorer, a periodontal probe, dry gauze sponges, and an air syringe. The need for specialized instrumentation and additional diagnostic procedures will vary with the findings and differential diagnoses developed.

Examine the Vermilion of the Lips

The mouth begins at the mucocutaneous junction of the vermilion border of the lips. The vermilion border, a zone of specialized non-mucus-producing tissue, is bounded by the facial skin and the moist labial mucosa of the mouth. With age and exposure to the elements, the color of the vermilion border may change from a pink or red (vermilion) to a bluish hue. This region is a common site for physiological pigmentation, ephelides or freckles, and rarely, pigmentation suggestive of **Peutz-Jeghers syndrome**. Varices, hemangiomas, and rarely, telangiectasias (**hereditary hemorrhagic telangiectasia** and CREST syndrome) may be noted.

Swelling of the lip may indicate cellulitis of dental origin or **angioedema**. Vesicular lesions of the lip may represent the initial phase of recurrent herpes labialis (RHL) (see **"Herpetic Infections"**). Serohemorrhagic crusting of the lips is highly suggestive of **erythema multiforme**, an acute vesiculoulcerative disorder. Thickening of the vermilion border with the development of vertical fissures and/or the loss of distinction of the mucocutaneous junction may be indicative of **actinic cheilosis**. The upper and lower lips join at the commissures. Crusting or weeping erosion in this area is characteristic of **angular cheilitis**.

Peutz-Jeghers Syndrome

Peutz-Jeghers syndrome (PJS) is a rare autosomal dominant disorder characterized by mucocutaneous melanin pigmentation and unique hamartomatous polyps (Peutz-Jeghers polyps) affecting the gastrointestinal tract. These polyps have a predilection for the small intestine. Mutations of the *LKB1* tumor suppressor gene have been demonstrated in up to 80% of cases. PJS patients are at an increased risk of cancer, especially cancer of the colon, stomach, small intestine, pancreas, breast, and ovary. The incidence of PJS is estimated to be 1 in 50,000 to 1 in 200,000.

Clinical Features

The characteristic mucocutaneous pigmentation presents as 1–5 mm dark brown or blue macule affecting the lip vermilion (Figure 5.1a), buccal mucosa, hands (Figure 5.1b), and feet. These macules typically develop in infancy, but may fade with age. Since these areas of pigmentation are asymptomatic, their presence is often underappreciated. Underlying gastrointestinal symptoms, such as intestinal obstruction, abdominal pain, and bloody stools, often prompt the patient to seek medical attention.

Diagnosis

The diagnosis of PJS is straightforward and established by the presence of either two or more Peutz-Jeghers polyps, one Peutz-Jeghers polyp and mucocutaneous pigmentations, or one Peutz-Jeghers polyp and a positive family history for PJS.


a


b

Figures 5.1a and b. Peutz-Jeghers syndrome.

Treatment

There is no cure for PJS. The increased cancer risk associated with PJS mandates earlier and more frequent screening for malignant disease. The mucocutaneous pigmentation requires no treatment.

Hereditary Hemorrhagic Telangiectasia

Hereditary hemorrhagic telangiectasia (HHT) is an uncommon autosomal dominant fibrovascular disorder. Also known as Rendu-Osler-Weber disease, HHT is characterized by the triad of mucocutaneous telangiectasias, arteriovenous malformations, and recurrent hemorrhagic episodes. The morbidity and mortality of HHT is mainly attributable to the arteriovenous malformations (AVMs), which may occur in the lungs, skin, brain, liver, and gastrointestinal tract. Mutations underlying HHT have been identified in two different genes: edoglin (*ENG*) and activin-receptor-like-kinase (*ALK1*). For either case, homozygous forms of HHT are considered lethal. The incidence of HHT is estimated to be 1 in 5,000 to 1 in 8,000.

Clinical Features

The most common presenting feature of HHT is recurrent epistaxis, which usually occurs between the ages of 10 and 20 years and may progress in frequency and severity with age. The telangiectasias become apparent in the third to fourth decade of life and usually progress both in size and number with age. They present as small flat or slightly raised bright red-purple lesions. These lesions blanch upon pressure and may be found on the face (Figure 5.2a), conjunctivae, lips (Figure 5.2b), tongue, palate, gingivae (Figure 5.2c and 5.2d), fingers (Figure 5.2e), arms, and trunk. Gastrointestinal involvement may result in severe blood loss leading to severe anemia with pallor (Figure 5.2f). AVMs are associated with pulmonary shunting, transient ischemic attacks, brain abscess, and hepatic shunting.

Diagnosis

The diagnosis is based on the presence of three of the following four findings: spontaneous recurrent epistaxis, mucocutaneous telangiectasia, visceral involvement, and an affected first-degree relative.

Treatment

There is no cure for HHT, and current treatment strategies entail close monitoring to identify and treat a potentially lethal AVM. Symptomatic treatment of epistaxis is often required, as is iron supplementation to manage anemia.



Figures 5.2a–f. HHT.

Angioedema

Angioedema (AE) is a reversible localized swelling of the deep subcutaneous or submucosal tissues secondary to increased vascular permeability caused by locally produced vasoactive substances such as histamine, tryptase, prostaglandin $F_{2\alpha}$, and bradykinin. While there are many established causes for AE (Table 5.1), the cause in most cases remains unknown. The true incidence of AE is unknown, but it is postulated that up to 20% of the population will experience at least one episode of AE in a lifetime.

Table 5.1. Causes of angioedema.

Mechanism of disease	Precipitating factors
IgE mediated allergic response	Insect venom, shellfish, peanuts, antibiotics, NSAIDs
Pseudoallergic response	Opioids, polymyxin, ACE inhibitors
Autoimmune disease	Hashimoto thyroiditis
C1 esterase inhibitor (C1-INH) deficiency	Hereditary, acquired, idiopathic
Trauma	Mechanical, chemical, thermal

Clinical Features

While any part of the body may be affected, most cases are characterized by edema of the face and/or pharyngeal tissues (Figures 5.3a and 5.3b). The affected skin or mucosa may be tender and warm and the patient may complain of a burning sensation. Severe AE affecting the head and neck may spread to the larynx, resulting in life-threatening airway obstruction. About 50% of patients also manifest urticaria and pruritis. Involvement of the abdominal viscera is often associated with severe pain.

Diagnosis

The abrupt onset of AE affecting an observable area is easily recognized and diagnosed.

Treatment

The initial treatment often focuses on providing symptomatic relief. AE presenting with urticaria usually responds well to antihistamines and corticosteroids. Efforts to identify the cause of the AE should be undertaken, and patients with recurrent AE should be referred to an allergist or dermatologist for a comprehensive evaluation.

a 

 b

Figures 5.3a and b. Angioedema.

Erythema Multiforme

Erythema multiforme (EM) is an acute, typically self-limiting, and potentially recurrent mucocutaneous vesiculoerosive disorder. Severity varies from mild (EM minor) to moderate (EM major) to potentially fatal (Stevens-Johnson syndrome [SJS] and toxic epidermal necrolysis [TEN]) (Table 5.2). Some authorities consider oral EM minor/EM major and SJS/TEN to be two distinct disease processes.

The etiology of EM is most likely due to a genetically predisposed allergic host response to antigenic challenge (Table 5.3). Most cases of EM minor and EM major are related to an infectious agent, typically herpes simplex virus, while most cases of SJS and TEN are related to a pharmacologic agent, most frequently a sulfonamide, an anticonvulsive drug, or a COX-1 inhibitor. In many cases the causative agent is not identified. The incidence of EM minor and EM major is unknown, while the incidence of SJS and TEN is estimated to be two to three cases per million.

Clinical Features

Cutaneous lesions usually begin as erythematous papules that progress to form the more characteristic iris or target lesions. These lesions typically arise in an acral distribution (Figure 5.4a). In more severe forms of EM, the cutaneous lesions may present as more widespread erythematous or purpuric macules and blisters. Such patients may demonstrate a positive Nikolsky's sign. Hemorrhagic crusting of the lips is highly characteristic

Table 5.3. Some causes of erythema multiforme.

Infectious agents	Drugs
β-Hemolytic streptococci	Barbiturates
Coccidiomycosis	Carbamazepine
Coxsackie virus	Cephalosporins
Diptheria	Clotrimazole
Epstein-Barr virus	Fluoquinolones
Herpes simplex 1 & 2	NSAIDs
Herpes zoster	Penicillin
Influenza, type A	Phenytoin
Mumps	
Mycoplasma pneumoniae	
Vaccinia	

Table 5.2. Spectrum of erythema multiforme.

Type	Cutaneous involvement	Mucosal involvement	Outcome
EM minor	Target lesion, acral distribution, negative Nikolsky's sign	Often absent	Recovery; possible recurrence
EM major	As above	Prominent oral involvement; vesiculoerosive erosions with fibrinous pseudomembrane; characteristic hemorrhagic lip involvement	Recovery; possible recurrence; rare mortality
SJS	Widespread small blisters, macules, atypical target lesions predominate on torso; epidermal detachment <10% body surface area; positive Nikolsky's sign	As above, possibly more extensive; ocular and genital involvement common	Fatal in 5–10% of cases; possible scarring; possible recurrence
TEN	Widespread small blisters, macules, atypical target lesions predominate on torso; epidermal detachment in >30% body surface area; positive Nikolsky's sign	As above	Fatal in up to 35% of cases; possible scarring; possible recurrence
Oral EM	Typical target lesions frequently absent	Oral lesions predominate clinical picture	Recovery; possible recurrent and chronic forms



Figures 5.4a–d. Erythema multiforme.

and virtually pathognomonic (Figure 5.4b). Intraorally, lesions on the unattached mucosal tissues predominate (Figure 5.4c and 5.4d). In the vast majority of cases, mucosal lesions tend to appear abruptly and manifest as painful vesicles, ulcerations, and erosions. Mucocutaneous lesions tend to heal completely in 2–6 weeks.

Diagnosis

The typical abrupt onset, combined with the presence of the characteristic mucocutaneous lesions (i.e., target lesions, crusted lips, and vesiculoulcerative oral lesions) is diagnostic. Historical evidence of prior occurrence and/or exposure to a possible causative drug or infectious agent reinforces the diagnosis. For equivocal cases, a biopsy and immunofluoresence studies may be useful to rule out other conditions in the differential diagnosis. Conditions that may mimic EM include erosive lichen planus, pemphigus, mucous membrane pemphigoid, lupus erythematosus, herpetic gingivostomatitis, major aphthous, and hand-foot-and-mouth disease.

Treatment

Most cases of EM resolve in 2–6 weeks, and treatment is generally palliative and supportive. Efforts to ensure adequate hydration and nutrition are mandatory, as is close monitoring. Anesthetic mouth rinses such as diphenhydramine hydrochloride and viscous lidocaine may be prescribed for oral pain, along with a bland soft nutritious diet. Antibiotic therapy may be indicated if secondary infection becomes evident. The withdrawal of any suspected causative medication should be undertaken and a careful history should be obtained to identify other possible underlying causes. Suspected cases of SJS or TEN mandate immediate referral to a physician.

Actinic Cheilosis

Actinic cheilosis is essentially actinic keratosis affecting the lip vermilion. Etiologic factors include cumulative ultraviolet radiation (UVR) exposure, skin phenotype, age, male sex, outdoor occupation, tobacco habits, and host immunological status. It represents the early clinical stage of a continuum that ultimately may progress to squamous cell carcinoma of the lip. Cumulative exposure to UVR in sunlight is the most important cause of actinic cheilosis. The true incidence of actinic cheilosis is unknown.

Clinical Features

Actinic cheilosis typically develops over several years and primarily affects the lower lip. Initially, the patient may manifest a dry, unobtrusive chapped lip (Figure 5.5a). More advanced cases manifest marked parallel folds, isolated hyperkeratotic plaques, and a loss of the definition of the vermilion-cutaneous border (Figure 5.5b). In later stages, actinic cheilosis may appear mottled or opalescent in color, with a slightly elevated white or gray plaque (Figure 5.5c). The waxing and waning of erythematous or hemorrhagic areas over a prolonged period of time may represent an ominous sign.

Diagnosis

The working diagnosis of actinic cheilosis usually is straightforward and is derived by correlating the history with clinical findings in an at-risk patient. The presence of concurrent actinic keratoses on sun-exposed areas (i.e., face, neck, bald scalp, and ears) reinforces the diagnosis. Lesions that persist or do not respond to preventive measures should be biopsied. Unfortunately, the clinical appearance of actinic cheilosis does not always correlate directly with the underlying histological changes. Suspicious-looking lesions may prove to be remarkably benign, while a small area of actinic cheilosis may in fact represent severe dysplasia or even squamous cell carcinoma.


a


b


c

Figures 5.5a–c. Actinic cheilosis.

Treatment

Commonly attempted ablative therapies to remove actinic cheiloses include cryotherapy with liquid nitrogen, topical 5-fluorouracil (5-FU), excision, laser ablation, and chemical peel. Given the strong etiologic link between UVR and actinic cheilosis, reducing exposure to sunlight (or other forms of UVR) is the single most important measure for preventing actinic cheilosis. Avoiding peak sun exposure; covering up exposed skin; wearing a hat that shades the neck, face, and ears; wearing sunglasses; and using a sunscreen with a sun protection factor (SPF) of 15 or higher is recommended for all patients.

Angular Cheilitis

Angular cheilitis (perlèche) is an erythematous fissuring of the commissures of the mouth and adjacent skin. The lesions are typically infected with *Candida albicans* and staphylococci or streptococci. Factors that predispose to the development of angular cheilitis include insufficient vertical dimension of occlusion, nutritional deficiencies, endocrinopathies, medications, poor oral hygiene, dental prostheses, and conditions of altered immunity. While angular cheilitis may be an isolated occurrence, it is often associated with intraoral candidiasis. The true incidence of angular cheilitis is unknown, but the number of patients who harbor *Candida albicans* as a commensal organism in the oral cavity varies from 25% to 75%.

Clinical Features

Angular cheilitis presents as unilateral (Figure 5.6) or bilateral (Figure 5.7) erythematous fissures of the commissures of the lips and adjacent skin. The lesion often has a glistening or moist appearance, but variable crusting may also occur.



Figure 5.6. Angular cheilitis.



Figure 5.7. Angular cheilitis.

Diagnosis

The characteristic site-specific presentation of angular cheilitis makes for a straightforward diagnosis.

Treatment

Mild cases of angular cheilitis are often transient and resolve uneventfully. More persistent cases typically respond to the use of a topical antifungal/steroid formulation and proper hygiene. For all cases, it is prudent to identify and, when possible, correct any predisposing factors.

Examine the Labial and Buccal Mucosa

To expose the labial mucosa, retract the upper and lower lips. Numerous minor salivary glands are located in this area. Using bidigital palpation, these glands are often apparent as small nodules. Minor salivary glands express saliva through pinpoint ducts. The function of these glands may be evaluated by everting the lips and drying the labial surfaces. Within several seconds, small beads of saliva should be expressed from these ducts. **Mucoceles**, extravasations of mucous-type saliva (mucin) into the connective tissue, occur frequently in the lower lip.

Still using the fingers as retractors, extend the examination to the buccal and vestibular mucosa. Observe color and other tissue characteristics. Find the parotid duct (Stensen's duct) located on the buccal mucosa adjacent to the maxillary first and second molar teeth. Assess parotid function. Firmly massaging the gland toward Stensen's duct should yield a clear fluid. Expression of casts, pus, or blood may indicate the presence of an infection, a sialolith, or a malignancy. Reduced salivary flow, **dry mouth** or xerostomia, is most often noted secondary to the use of anticholinergic agents, or in association with diabetes mellitus, Sjögren's syndrome, or head-and-neck radiotherapy.

A common and totally benign finding, linea alba typically presents bilaterally as a grayish-white keratotic linear plaque on the buccal mucosa that runs parallel to the adjacent occlusal plane. It is thought to represent mild occlusal trauma to the buccal mucosa characterized by hyperorthokeratosis (hyperkeratosis without retention of nuclei). More pronounced lesions may exhibit a shredded or shaggy appearance. Such pronounced cases likely indicate that a factitial habit (e.g., habitual cheek chewing) has contributed to the development of the lesion. Treatment is neither required nor recommended for linea alba.

In **leukoedema,** a normal mucosal variation, the mucosa appears wrinkled and opalescent. Traumatic lesions and **recurrent aphthous stomatitis** are commonly observed on the labial and buccal mucosa. The buccal mucosa is also a common site for the lacy linear Wickham striae of **lichen planus** and the diffuse thickened white lesions of **white sponge nevus**, a congenital condition. **Snuff keratosis** is most commonly observed in the mucobuccal fold.

Pigmented lesions affecting the oral tissues are rare. Fordyce granules (ectopic sebaceous glands) appear as clusters of small yellow nodules and are frequently observed in the labial and buccal mucosa. An **amalgam tattoo** and a hematoma may be observed as an isolated bluish or grayish pigmentation affecting almost any area of the mouth. Examination of the buccal mucosa may also reveal commonly occurring raised or nodular lesions such as a **fibroma** and a **papilloma,** or less commonly occurring lesions such as a neuroma, hemangioma, or malignant neoplasm. Other conditions that may affect the labial and buccal mucosa include erythema multiforme and **pemphigus vulgaris**.

Mucocele

A mucocele (mucous retention phenomenon, mucous extravasation phenomenon) represents a collection of salivary mucin entrapped within the connective tissue. It is thought that microtrauma to a minor salivary gland causes ductal disruption followed by mucin extravasation into the surrounding soft tissue. They are quite common, demonstrate no sex predilection, and occur more frequently in children, adolescents, and young adults. Mucoceles are most frequently located in the lower lip (60–70%), but may develop at any location where minor salivary glands are present, including the soft palate, retromolar region, and buccal mucosa. Their occurrence in the maxillary lip is uncommon.





Figures 5.8a and b. Mucocele.

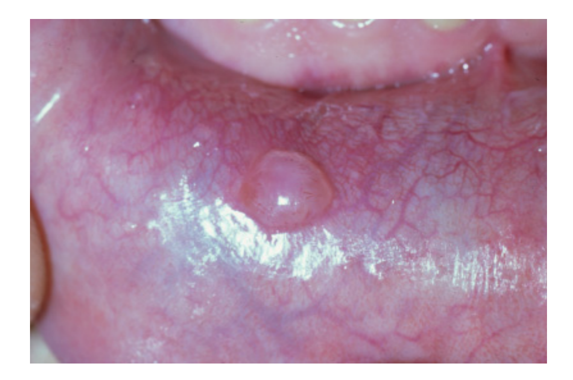


Figure 5.9. Mucocele.

Clinical Features

While some mucoceles may become large enough to cause a visible lip swelling (Figures 5.8a and 5.8b), typically they appear as a discrete, small, translucent, soft, painless swelling of the mucosa (Figure 5.9). Their color may range from normal pink to deep blue. A deep blue color reflects tissue cyanosis as a result of vascular congestion associated with the stretched overlying tissue and the translucent character of the accumulated fluid beneath. A more deep-seated lesion may appear as a normal colored submucosal nodule.

Diagnosis

The diagnosis of a mucocele is based principally on clinical and historical findings. The patient may or may not recall a specific inciting traumatic event. Lesions that may mimic a mucocele include a fibroma, vascular lesion, neural tumor, or salivary gland tumor.

Treatment

Smaller mucoceles often undergo spontaneous involution and resolution. Lesions that persist or interfere with normal function are easily managed surgically. It is important that the surgeon remove all visible minor salivary gland tissue at the time of surgery, in order to reduce the risk of recurrence.

Dry Mouth

Dry mouth is a commonly encountered problem in dental practice. The term used to describe the patient's subjective complaint of a dry mouth is xerostomia. Numerous etiologic factors such as salivary gland disease, systemic disease, medications, head and neck irradiation, and some chemotherapy regimens contribute to dry mouth. Xerostomia is a common presenting complaint in about 30% of patients over the age of 65 years.

Clinical Features

The oral findings associated with dry mouth are variable and depend on the severity and

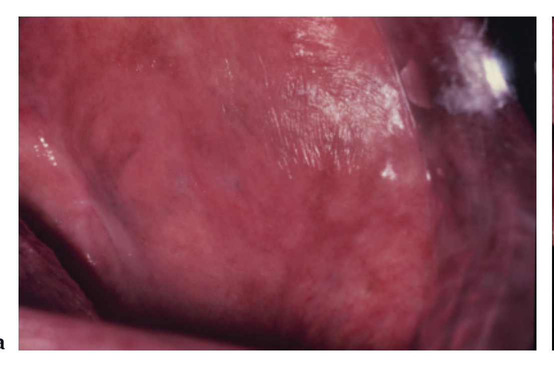
duration of dryness. Mild acute cases may manifest no overt clinical changes. Common findings of oral dryness include a lack of saliva pooling in the floor of the mouth; inadequate wetting of the mucosal tissues (Figure 5.10a); a dry, furrowed, or fissured tongue (Figure 5.10b); patchy areas of erythema, usually indicative of candidiasis; and rampant caries (Figures 5.11a and 5.11b).

Diagnosis

The diagnosis of a dry mouth is relatively straightforward; the challenge is to identify the underlying cause or causes. Obtaining an exhaustive medical history is mandatory, and further medical evaluation may be necessary.

Treatment

Treatment strategies for dry mouth are individually tailored and largely contingent upon the severity and duration of the dry mouth. Medication adjustment and/or underlying disease management may be all that is necessary to reestablish normal salivary production. Episodic cases, such as may occur with occasional allergy medication ingestion, may be managed with simple supportive measures such as frequent sipping of water throughout the day. In contrast, the more severe and permanent dry mouth associated with head and neck irradiation is often only minimally responsive to currently available management protocols. These protocols consist of improved oral hygiene, saliva sub-

a 

 b

Figures 5.10a and b. Xerostomia.

a 

 b

Figures 5.11a and b. Xerostomia.

stitutes, fluoride therapy, and sialogogue administration.

Leukoedema

Leukoedema is a common normal variation of the buccal mucosa. The etiology is unknown, but some authorities suggest low-grade irritation contributes to an increased thickness of the epithelium, intracellular edema in the prickle cell layer, and hyperparakeratosis (hyperkeratosis with retention of nuclei). Prevalence rates vary greatly among geographic locations and among different ethnic groups. It may be present at birth, but is most often diagnosed in adolescence. Prevalence rates in blacks vary from 70% to 90%, while rates in whites vary from 10% to 90%.

Clinical Features

Leukoedema presents as a bilateral white, filmy, or milky opalescence of varying intensity affecting the buccal mucosa (Figure 5.12). The mucosa may appear folded or wrinkled (Figure 5.13). Leukoedema either disappears or undergoes significant diminution when the affected tissue is stretched. Infrequently, leukoedema affects the tongue, lips, and floor of the mouth and similar mucosal changes have been reported affecting vaginal and laryngeal mucosa.

Diagnosis

The tell-tale disappearance upon stretching is diagnostic. Other white lesions that may mimic leukoedema include white spongy nevus, snuff keratosis, lichen planus, proliferative verrucous leukoplakia, and hereditary benign intraepithelial dyskeratosis (Witkop disease).

Treatment

No treatment is necessary for leukoedema. It has no malignant potential and does not change significantly after 25–30 years of age.



Figure 5.12. Leukoedema.



Figure 5.13. Leukoedema.

Recurrent Aphthous Stomatitis

"Aphthous" comes from the Greek word "aphtha," which means ulcer. Recurrent aphthous stomatitis (RAS) is a recurrent, self-limiting, noninfectious inflammatory disorder of the oral cavity characterized by painful shallow ulcerations. The actual cause of RAS remains unclear, although it is a T-cell-mediated condition and a genetic predisposition is likely. RAS is considered the most prevalent oral mucosal disease, affecting an estimated 20% of the population, and has been associated with numerous other conditions (Table 5.4). "Aphthous stomatitis" has been used interchangeably with "aphthous ulcers" and may be more accurate terminology.

Clinical Features

RAS typically presents as one or more well-circumscribed, round or oval, yellowish to white colored, shallow ulcers that are surrounded by an erythematous halo of inflamed mucosa. The lesions are typically painful and may interfere with speaking, eating, and swallowing. Some patients relate a prodromal burning sensation 24–48 hours before lesion onset. RAS characteristically affects the nonkeratinized oral mucosa, such as the labial and buccal mucosa (Figures 5.14, 5.15, and 5.16), tongue (Figure 5.17), and soft palate. RAS may be further characterized

Table 5.4. Conditions often associated with RAS.

Local trauma (factitial or iatrogenic)	Hematological diseases (cyclic neutropenia, leukemia)
Stress	Rheumatic disorders (Behçet disease, Reiter's syndrome)
HIV infection	Allergy (food, drugs)
GI diseases (Crohn's disease, ulcerative colitis, and celiac disease)	Nutritional deficiencies (iron, folic acid, zinc, and vitamins B_1, B_2, B_6, and B_{12})

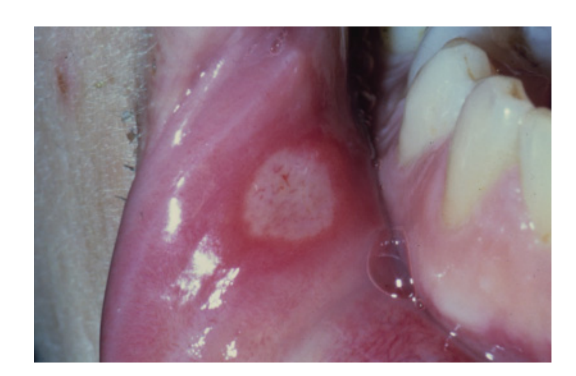


Figure 5.14. RAS.



Figure 5.15. RAS.

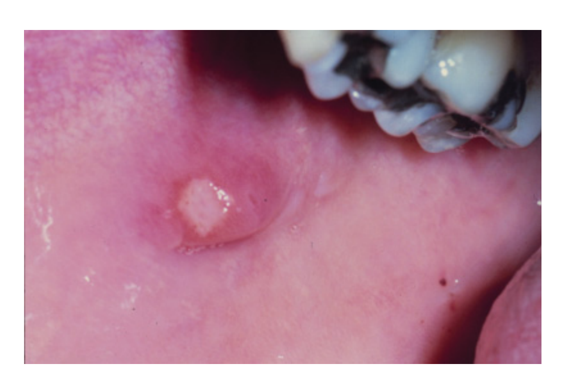


Figure 5.16. RAS.

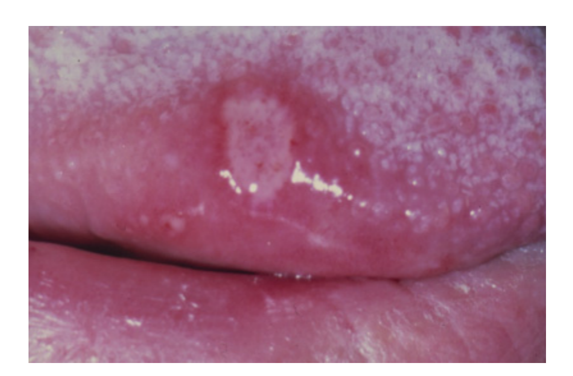


Figure 5.17. RAS.

Table 5.5. Classification of RAS.

Type	Characteristics
Minor RAS	Most common type (80%); single or multiple small ulcers (< 1.0 cm); heal without scarring in 7–10 days.
Major RAS	Less common type (10–15%); single or multiple large ulcers (> 1.0 cm); ulcers are deeper and more persistent; heal in several weeks; scarring possible.
Herpetiform RAS	Least common (5–10%), multiple small pinpoint ulcerations; clustered or cropped presentation; heal in 1–4 weeks.

according to its clinical appearance and severity (Table 5.5).

Diagnosis

The diagnosis of RAS is principally established based upon the characteristic clinical presentation and history. Conditions to consider in the differential diagnosis include recurrent intraoral herpes simplex infections, primary herpetic gingivostomatitis, herpes zoster, a traumatic ulcer, EM, and ulcers associated with systemic disease.

Treatment

The goals of therapy are to relieve pain, promote healing, and decrease the frequency and severity of recurrence. Although a variety of treatment modalities (topical and systemic) have been promoted to either eliminate or reduce the duration of recurrence of RAS, their clinical value remains unproven and can be controversial. On the basis of efficacy, cost, and safety, topical steroids remain the treatment of choice for patients with minor RAS. Finally, a comprehensive medical evaluation may be warranted to identify underlying systemic conditions potentially associated with RAS.

Lichen Planus

Lichen planus (LP) is a cell-mediated chronic inflammatory disease involving mucous membranes and skin. It is thought to arise as a result of an immune response to certain unknown antigens within the basal cell layer of the epithelium. LP primarily occurs in middle-aged individuals, although all ages are at risk. The disease has a predilection for women (2 : 1). Up to 65% of patients with cutaneous LP will manifest concurrent oral lichen planus (OLP), and oral lesions comprise the sole manifestation of LP in approximately 15–35% of cases. OLP affects an estimated 0.1–4% of the population.

Clinical Features

There are at least three recognized forms of OLP: reticular, atrophic, and erosive. The reticular variant is thought to be the most common form and is characterized by mucosal keratotic lines, plaques, or papules arranged in a characteristic lacy pattern of Wickham striae (Figures 5.18a–5.18f). The atrophic form is a combination of the reticular form and erythematous component (Figure 5.19), while the erosive form is a combination of the reticular form and a shallow ulcerative component (Figure 5.20a). All forms of LP may be accompanied by skin lesions (Figure 5.20b). Reticular lesions tend to be asymptomatic, while atrophic and erosive forms are likely to be painful. The most commonly affected sites are the buccal mucosa, tongue, lips, floor of the mouth, palate, and gingivae. OLP typically presents in a bilateral and symmetrical fashion. Patients often present with a variable mix of all three forms, and in some cases the characteristic striae may be absent. OLP may wax and wane, but seldom undergoes total remission.



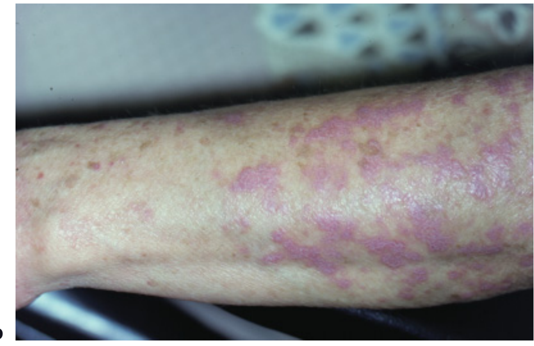
Figures 5.18a–f. Lichen planus.



Figure 5.19. Lichen planus.



a



b

Figures 5.20a and b. Lichen planus.

Diagnosis

A working diagnosis of LP is usually established when the characteristic striae are present. A confirmatory biopsy should always be obtained and is essential in order to diagnose equivocal cases. The variable appearance of OLP results in an extensive differential diagnosis including oral candidiasis, lichenoid drug reactions, erythema multiforme, epithelial dysplasia, mucous membrane pemphigoid, pemphigus vulgaris, oral cancer, periodontal disease, graft-versus-host disease, lupus erythematosus, gastrointestinal disease, anemia, leukoplakia, erythroplakia, and linear IgA disease.

Treatment

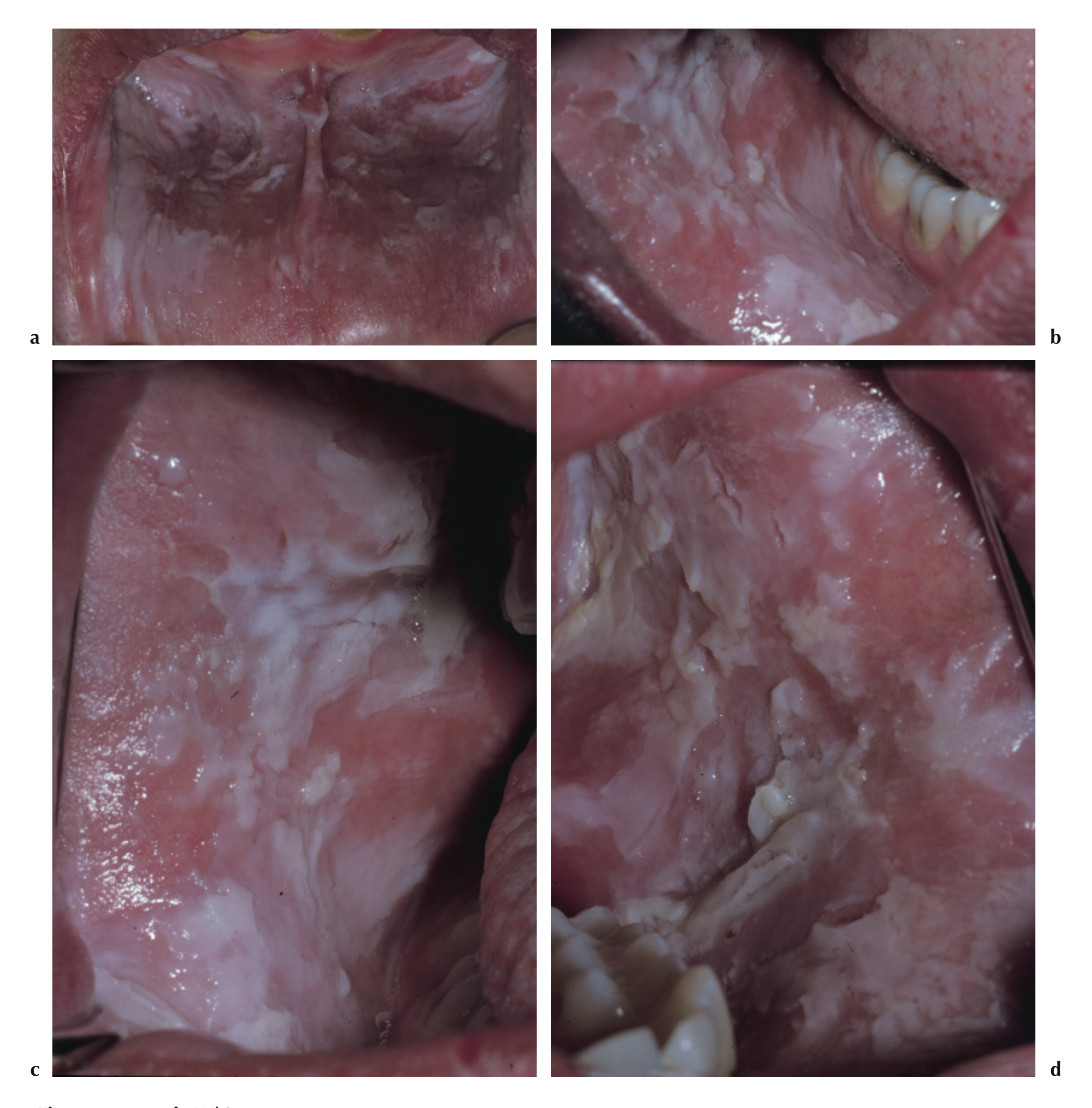
There is no cure for OLP, and treatment is based on symptoms. Asymptomatic reticular OLP requires no treatment. Symptomatic atrophic or erosive forms of OLP often respond well to topical steroids, while more severe disease may require systemic therapy. In all cases, OLP should be routinely monitored, as patients with OLP appear to be at an increased risk of developing oral cancer.

White Sponge Nevus

White sponge nevus is an uncommon benign hereditary disorder that affects the mucous membranes.

Clinical Features

White sponge nevus is characterized by the presence of folded or corrugated white plaques. Symmetrical involvement of the buccal mucosa is invariably observed (Figures 5.21a–5.21d.), but other oral mucosal sites, along with the nasal, vaginal, and rectal mucosa, may be affected.



Figures 5.21a–d. White sponge nevus.

Diagnosis

The clinical appearance, combined with a family history of disease, makes for an easy diagnosis.

Treatment

No treatment is necessary for this benign condition.

Snuff Keratosis

Snuff keratosis represents the reactive mucosal response to the placement of smokeless tobacco products. The smokeless tobacco, along with numerous other ingredients within the smokeless tobacco, acts to irritate the oral mucosal membranes. While there is a tangible risk of dyplasia occurring in snuff keratosis, recent epidemiological data suggests the prevalence of oral epithelial dysplasia in such lesions is generally low. While the rate of malignant transformation is low and the process is slow, the relative risk for carcinoma of the buccal/labial mucosa and gingivae among female chronic users is 50 times greater than among nonusers. It is estimated that 7.7 million people used smokeless tobacco products in 2003.

Clinical Features

Snuff keratosis presents as a fairly characteristic corrugated or folded grayish-white plaque affecting the labial and vestibular mucosa (Figures 5.22, 5.23, 5.24, and 5.25). These lesions develop in those areas where the smokeless product is routinely placed. Most lesions are fairly well localized, but those associated with extensive smokeless tobacco use may be widespread.

Diagnosis

The clear association between smokeless tobacco placement and lesion development makes for a straightforward diagnosis.



Figure 5.23. Snuff keratosis.



Figure 5.24. Snuff keratosis.



Figure 5.22. Snuff keratosis.

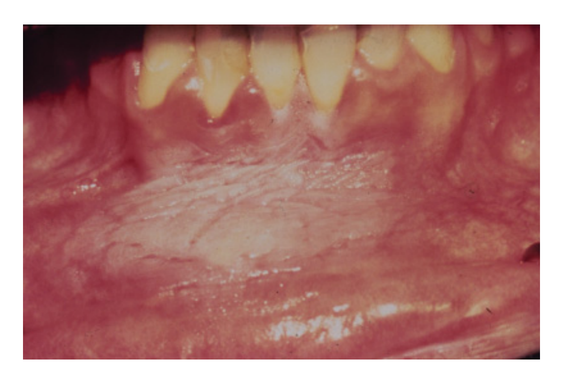


Figure 5.25. Snuff keratosis.

Treatment

Any patient with snuff keratosis should be encouraged to quit using smokeless tobacco and should be advised of available cessation resources. Discontinuation of the smokeless tobacco usually results in lesion resolution. A biopsy is rarely necessary, but is indicated to assess any area of snuff keratosis that is clinically suspicious or unusual looking.

Amalgam Tattoo

An amalgam tattoo is a well-circumscribed, flat pigmented oral lesion. They arise when small particles of dental amalgam, composed primarily of silver (Ag) and mercury (Hg), are inadvertently implanted into the oral soft tissues during dental procedures. It has also been suggested that they may develop in the oral soft tissues as a result of chronic exposure to restorative materials containing Hg, Ag, and sometimes copper, zinc, and tin. Amalgam tattoos are common, affecting about 8% of the population.

Clinical Features

An amalgam tattoo typically presents as an asymptomatic, solitary gray, blue, or black macule on the oral mucosa. They most commonly occur on the gingivae, alveolar ridge (Figure 5.26a), cheek, floor of the mouth, palate, and tongue. Most lesions are less than 1 cm in size. In some cases, evidence of small radiopaque amalgam flecks may be observed on a routine radiograph (Figure 5.26b).

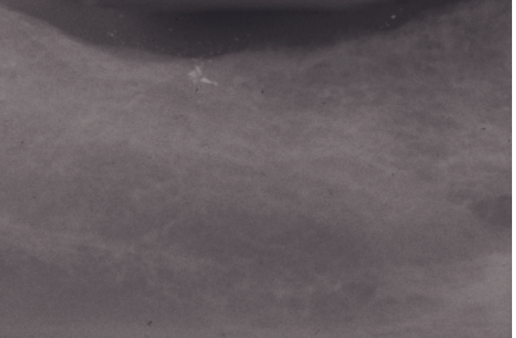
Diagnosis

The diagnosis of an amalgam tattoo is typically based on the characteristic clinical findings combined with the history of prior dental work. A biopsy is recommended for equivocal cases. Other lesions to consider in the differential diagnosis include physiologic pigmentation, an oral melanotic macule, nevi, melanoma, Kaposi's sarcoma, pigmentation associated with systemic disease (e.g., Addison's disease, Cushing's syndrome), and disorders related to blood or blood vessels.

Treatment

An amalgam tattoo does not require treatment. However, all pigmented oral lesions should be viewed with suspicion and if the diagnosis is questionable, a biopsy should be performed. For lesions deemed cosmetically unacceptable, surgical or laser ablation may be accomplished.


a


b

Figures 5.26a and b. Amalgam tattoo.

Fibroma

A fibroma is best considered a reactive hyperplasia of fibrous connective tissue in response to local irritation. The most common sites of occurrence correspond well with areas prone to intraoral trauma. The fibroma is accepted as the most common soft-tissue tumor affecting the oral cavity.

Clinical Features

A fibroma typically presents as a well-defined, smooth-surfaced nodule on the buccal and labial mucosa, gingivae, and tongue (Figures 5.27 and 5.28). The color ranges from pink to white. It is a slow-growing lesion that rarely exceeds 2 cm in size. Most examples have a sessile base, but some are pedunculated. A fibroma may demonstrate evidence of secondary trauma, such as surface ulceration or maceration.



Figure 5.27. Fibroma.



Figure 5.28. Fibroma.

Diagnosis

A recent episode of trauma to a pre-existing fibroma often prompts the patient to seek care. The rather banal appearance, combined with a history of trauma, makes for a straightforward initial diagnosis. A biopsy is required to establish the definitive diagnosis. Lesions to consider in the differential diagnosis include a neural tumor and a lipoma.

Treatment

Simple surgical excision is the treatment of choice and recurrence is rare.

Papilloma

A papilloma is a benign exophytic growth of the epithelium. The human papillomavirus (HPV), of which there are over 100 subtypes, is responsible for numerous conditions ranging from the common wart to more serious conditions such as genital warts and cancer. Collectively, an estimated 20 million Americans have been infected with HPV. Prevalence rates as high as 4.6 per 1,000 have been reported.

Clinical Features

The papilloma typically presents as a solitary smooth, papillomatous, or cauliflower-like projection of the epithelium (Figures 5.29, 5.30, 5.31, and 5.32). Oral papillomas most commonly affect the palate, uvula, tongue, gingivae, and buccal mucosa. The color ranges from pink to white and pedunculation is frequently observed.

Diagnosis

The characteristic clinical appearance usually allows for a straightforward diagnosis.



Figure 5.29. Papilloma.



Figure 5.30. Papilloma.

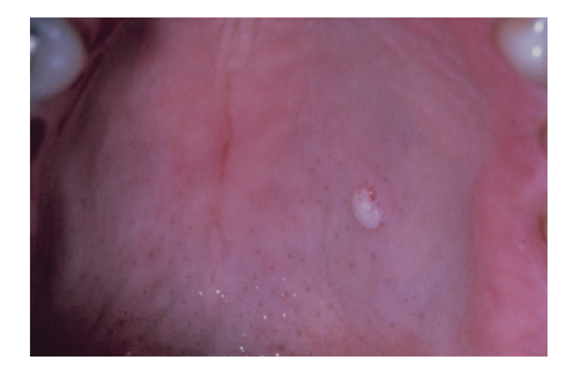


Figure 5.31. Papilloma.

However, a biopsy is recommended to confirm the diagnosis. HPV subtyping is available but is rarely used for establishing a diagnosis. A new onset of multiple papillomas should alert the clinician to the possibility of underlying immune compromise.



Figure 5.32. Papilloma.

Conditions to consider in the differential diagnosis include genital warts, focal epithelial hyperplasia, molluscum contagiosum, giant cell fibroma, and verruciform xanthoma.

Treatment

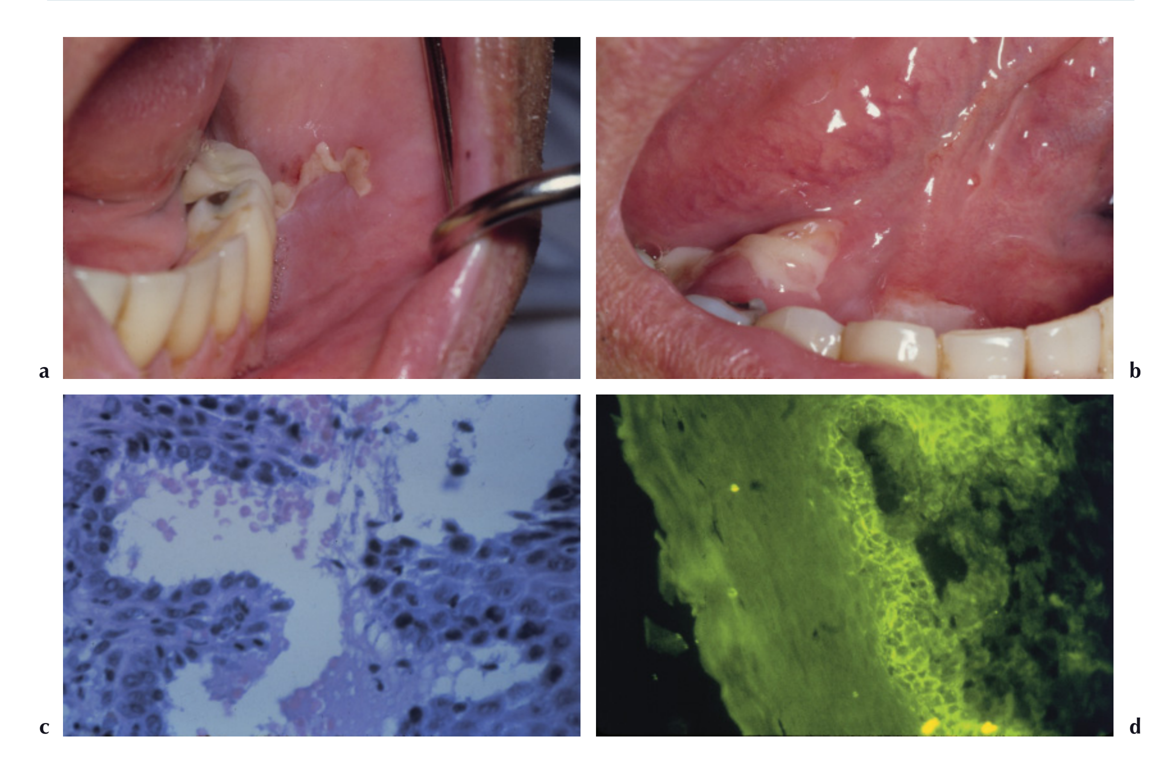
Simple excision of a solitary papilloma is the treatment of choice.

Pemphigus Vulgaris

Pemphigus vulgaris is a rare autoimmune disorder that affects the mucocutaneous tissues. Affected patients produce IgG autoantibodies that target the desmosomal adhesion molecules (i.e., desmoglein 1 and desmoglein 3) of the squamous epithelium. This destruction results in the development of an intraepithelial split. The mean age of onset is between 40 and 60 years of age and there is no sex predilection. An increased risk has been noted in persons of Mediterranean and Jewish descent.

Clinical Features

Pemphigus vulgaris typically presents as painful oral bullae, erosions, or ulcers (Figures 5.33a and 5.33b). These lesions usually develop insidiously and tend to persist. Often, a blister may be deliberately induced by



Figures 5.33a–d. Pemphigus vulgaris.

rubbing an unaffected area (Nikolsky's sign). The most commonly affected sites are the palate, buccal mucosa, and gingivae.

Diagnosis

The clinical appearance of pemphigus vulgaris is not specific. A biopsy and immunofluorescent studies are required to establish the presence of intraepithelial slits (Figures 5.33c) and the presence of autoantibodies (Figure 5.33d) associated with the disease. Numerous conditions may mimic pemphigus vulgaris and include mucous membrane pemphigoid, erythema multiforme, linear IgA disease, drug-induced reactions, and paraneoplastic pemphigus.

Treatment

Pemphigus vulgaris is a potentially fatal disease for which there is no cure. However, long-term disease control may be obtained with immunosuppressive therapy. For lesions restricted to the oral cavity, topical corticosteroid therapy may suffice to control the disease. Systemic immunosuppressive therapy is indicated to manage nonresponsive cases and such therapy is best accomplished by working closely with the patient's physician.

Examine the Hard Palate

With the patient's mouth wide open and the head tilted back, the palatal vault can be easily examined, both visually and tactilely. The incisive papilla overlies the incisive canal, and the palatal rugae present as transversely oriented palatal folds that converge toward the incisive papilla. An incisive canal

cyst may present as a fluctuant, midline, anterior palatal swelling. A spongy palatal swelling lateral to the midline likely indicates the presence of a periapical or periodontal abscess, or more rarely an expansile lesion such as a minor salivary gland tumor. The palatine raphe divides the palate down the midline and may be slightly elevated. A **palatal torus** presents as a hard, midline "swelling."

Nicotine stomatitis imparts a whitish-gray color to the mucosal tissues. The presence of diffuse palatal erythema or papillary hyperplasia affecting the denture-bearing mucosa is suggestive of atrophic **candidiasis**. Recurrent intraoral herpes (see **"Herpetic Infections"**) most frequently occurs on the hard palate and is characterized by multiple small vesicles, which coalesce, then rupture to form painful shallow ulcerations. Pigmented palatal lesions such as oral melanotic macules and nevi occur with some frequency, as do amalgam tattoos. Less commonly observed but far more serious lesions include malignant **melanoma** and **verrucous carcinoma**. In patients with immunosuppression (acquired or therapeutic), **Kaposi's sarcoma** may be observed.

Palatal Torus

A palatal torus is a common, benign, localized bony protuberance arising on the hard palate. It consists of mature dense cancellous bone covered by a rim of cortical bone of variable thickness. The etiology is unknown. A prevalence rate of 8.5 per 1,000 has been reported, with a predilection for females. Palatal tori are most frequently observed in young adults and middle-aged persons, and the occurrence increases with age, achieving a plateau by the third decade. Similar lesions arising on the lingual alveolar process of the mandible are termed mandibular tori. Exophytic bone growth on the facial or buccal surfaces of the alveolar processes is known as exostosis.

Clinical Features

The palatal torus presents as a bony hard exophytic mass on the palate (Figures 5.34, 5.35, and 5.36). The shape is variable and may be dome-shaped, spindle-shaped,



Figure 5.34. Torus palatinus.



Figure 5.35. Torus palatinus.



Figure 5.36. Torus palatinus.

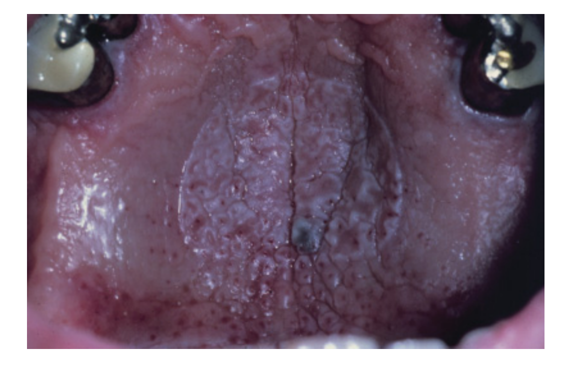


Figure 5.37. Nicotine stomatitis.

nodular, or lobular. The size varies from barely discernible to very large (1.5–4 cm in diameter). If a periapical radiograph of the affected area is taken, a focal area of increased density may be noted.

Diagnosis

The characteristic appearance of the palatal torus is pathognomonic. In some cases, the patient's first awareness of a torus occurs when the thin overlying mucosa becomes traumatized, prompting a visit to the dentist.

Treatment

A palatal torus rarely requires treatment. However, if it interferes with function, denture placement, or is repeatedly traumatized, the torus may be surgically removed.

Nicotine Stomatitis

Nicotine stomatitis is a specific red/white lesion attributable to smoking with relatively quick resolution after smoking cessation. Although it manifests no increase in malignant transformation, if the tobacco habit involves reverse smoking (i.e., placement of the lighted end in the mouth), then the palatal lesion should be deemed as precancerous.



Figure 5.38. Nicotine stomatitis.

Clinical Features

Nicotine stomatitis most frequently affects the hard and soft palate, followed by the retromolar pad and buccal mucosa. It produces a generalized thickening of the affected oral mucosa varying in appearance from a faint white-gray translucence to a distinct whitish-gray plaque (Figures 5.37 and 5.38). Palatal cases typically exhibit interspersed reddish puncta, which represent the dilated opening of minor salivary glands (Figures 5.39 and 5.40).

Diagnosis

The characteristic appearance, combined with a history of tobacco exposure, makes for a straightforward diagnosis. Unusual



Figure 5.39. Nicotine stomatitis.



Figure 5.40. Nicotine stomatitis.

appearing cases should be biopsied to establish a definitive diagnosis.

Treatment

Patients with nicotine stomatitis should be encouraged to stop using tobacco products, in which case the lesions may resolve significantly. Following discontinuation of tobacco products, the mucosa will eventually return to normal.

Candidiasis

Candidiasis is an infectious disease primarily caused by the mycelial form of *Candida albicans*. A normal inhabitant of the oral cavity, *Candida albicans* maintains a symbiotic relationship with *Lactobacillus acidophilus*. An alteration in host immune response likely precedes the metamorphosis from a state of commensalism to parasitism. Predisposing factors are summarized in Table 5.6.

Table 5.6. Predisposing factors for candidiasis.

Endocrinopathies (diabetes mellitus, hypoparathyroidism, hypoadrenalism, pregnancy)	Acquired and therapeutic immunosuppression (HIV infection, cytotoxic drugs, corticosteroids)
Nutritional deficiencies	Qualitative and quantitative changes in salivary flow (drug-induced, radiotherapy, Sjögren's syndrome)
High carbohydrate diet	Poor oral hygiene
Antibacterial agents	Dental prostheses
Age	Smoking

Considering the fact that carriage rates as high as 75% have been reported in healthy individuals, it is not surprising that candidiasis is a frequently encountered problem in clinical practice.

Clinical Features

Patients may be asymptomatic or may complain of burning, dysgeusia, and/or dysphagia. A variety of clinical forms have been proposed and described. Pseudomembraneous candidiasis is characterized by the presence of a "cottage cheese-like" pseudomembrane, which may be wiped away, leaving a painful, bleeding mucosal surface (Figures 5.41a–5.41d). This form is frequently observed in neonates and immunosuppressed patients. Erythematous candidiasis appears as red patches, most frequently affecting the palate or dorsum of the tongue (Figures 5.42, 5.43, and 5.44). This form may be associated with a dry mouth, an immunosuppressive state, or exposure to antibiotics. The least common form is hyperplastic candidiasis, which is characterized by

Figures 5.41a–d. Pseudomembraneous candidiasis.



Figure 5.42. Erythematous candidiasis.



Figure 5.43. Erythematous candidiasis.



Figure 5.44. Erythematous candidiasis.

the presence of persistent, white plaques that do not wipe off (Figures 5.45a, 5.45b, 5.46, and 5.47). This form is usually associated with immunosuppression. Denture stomatitis presents as an erythematous area beneath a denture-bearing surface (Figure 5.48). This form is frequently asymptomatic and associated with poor oral hygiene and/or continual wearing of a dental prosthesis. Median rhomboid glossitis is a candidal infection that affects the midline of the dorsum of the tongue and alters the normal appearance of the papilla in this location (Figures 5.49 and 5.50). Finally, candidiasis may also present as angular cheilitis, which has been previously described.


a


b

Figures 5.45a and b. Hyperplastic candidiasis.

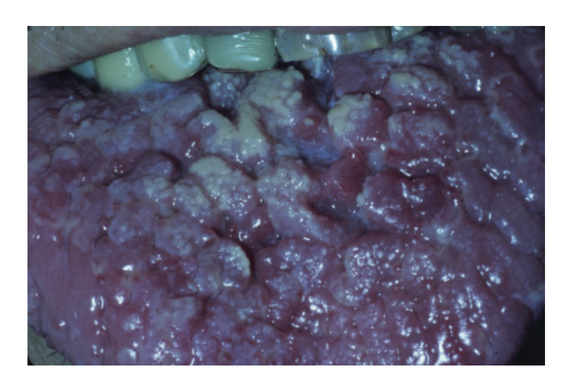


Figure 5.46. Hyperplastic candidiasis.

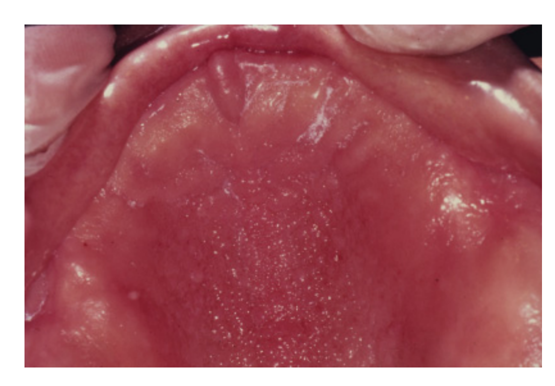


Figure 5.48. Denture stomatitis.



Figure 5.47. Hyperplastic candidiasis.



Figure 5.49. Median rhomboid glossitis.



Figure 5.50. Median rhomboid glossitis.

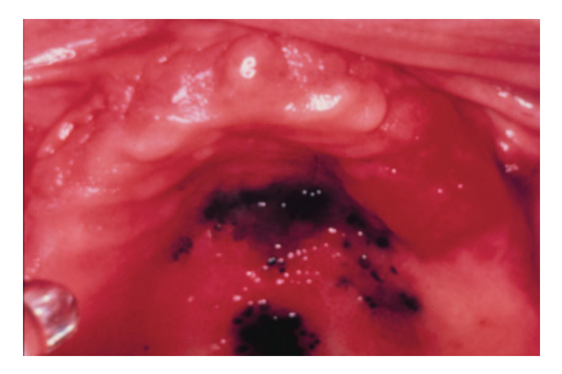


Figure 5.51. Melanoma.

Diagnosis

The diagnosis of candidiasis is established by correlating the typical signs and symptoms with the medical history. When doubt exists, exfoliative cytology, culture, or tissue biopsy may be used to confirm the diagnosis.

Treatment

Measures to address any predisposing factors should be accomplished. When necessary, anti-fungal agents, both topical and systemic, are available and should be individualized based on the patient's health status and the clinical presentation and severity of infection.

Melanoma

Melanoma is a rare entity. Compared with its cutaneous counterpart, oral melanoma tends to follow a more rapid clinical course, and the overall prognosis is very poor. An estimated 33–50% of all oral melanomas are believed to arise in an area of pre-existing pigmentation.

Clinical Features

The most common oral presentation is that of an expansile pigmented lesion most commonly affecting the palate and gingival mucosa (Figure 5.51). The color of the lesion may be pink, or it may contain any combination of blue, black, red, yellow, brown, and purple. Tooth mobility, spontaneous hemorrhage, satellite lesions, and pain may also be present.

Diagnosis

A biopsy is required for the diagnosis. The grave nature of oral melanoma, and its frequent association with a pre-existing area of pigmentation, dictates that all intraoral pigmented lesions be carefully assessed.

Treatment

Radical surgical resection is the treatment of choice. Additional chemotherapy, radiotherapy, and immunotherapy may be attempted, but the prognosis is very poor.

Verrucous Carcinoma

Verrucous carcinoma is an uncommon variant of squamous cell carcinoma that has a distinct predilection for the oral cavity. Compared with a typical squamous cell carcinoma, it tends to occur later in life (seventh to eight decade), is locally aggressive, and infrequently undergoes metastasis. Predisposing factors include oral tobacco use, smoking, alcohol, and poor oral hygiene.



Figure 5.52. Verrucous carcinoma.

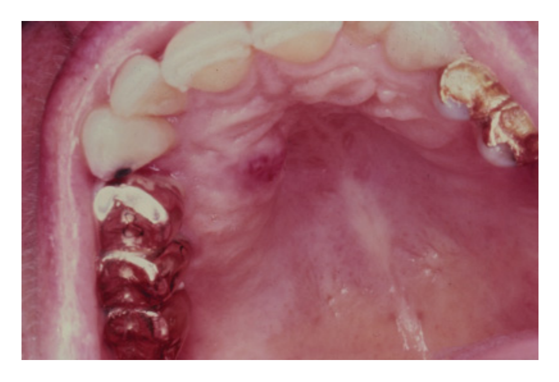


Figure 5.53. Kaposi's sarcoma.

Clinical Features

Verrucous carcinoma appears clinically as a prolific warty or fungating superficial growth (Figure 5.52). The most commonly affected sites are the buccal mucosa, gingivae, palate, and larynx. Areas of ulceration may be present.

Diagnosis

The characteristic clinical appearance should raise suspicion. A biopsy is warranted and should include ample sampling of the base of the lesion.

Treatment

Surgical excision is the treatment of choice, but is complicated by the tendency of the tumor to extend locally and to recur.

Kaposi's Sarcoma

Kaposi's sarcoma (KS) is an angioproliferative malignancy. It is the most common AIDS-associated malignancy, and its occurrence is closely associated with the sexual transmission of human herpesvirus type 8. While KS may occur anywhere in the body, up to 70% of initially observed lesions occur in the oral cavity.

Clinical Features

The most common sites of occurrence are the hard palate, gingival mucosa, and tongue (Figures 5.53 and 5.54a–5.54c). Initial lesions present as small red to blue macules, which progress to bluish expansile nodular masses of varying size. Advanced lesions may be painful and interfere with normal function.

Diagnosis

Advanced lesions observed in an HIV positive patient are difficult to miss clinically. However, early macular lesions occurring in patients unaware of their immune status are more problematic. In all cases a biopsy is necessary to confirm the diagnosis. Other conditions to consider in the differential include benign vascular lesions, trauma, and melanocytic lesions.

Treatment

Treatment of KS is palliative and targeted to alleviate pain and restore normal function. Potential therapeutic interventions include surgical excision or debulking, radiotherapy, and antineoplastic chemotherapy.



Figures 5.54a–c. Kaposi's sarcoma.

Examine the Soft Palate and Tonsillar Area

By gently depressing the base of the tongue, the soft palate and uvula can be directly examined. The soft palate functions to separate the oral cavity from the nasal pharynx and to close the nasal pharynx during swallowing and speaking. In contrast to the thick attached mucosa of the hard palate, which overlies bone, the mucosa of the soft palate is much thinner. Thus, it often appears pinker than the mucosa of the hard palate. In some patients, the underlying fat in the soft palate may impart a yellowish cast to the mucosa.

Petechia, purpura, and ecchymosis represent extravasations of blood into the connective tissue. Such lesions frequently occur in the soft palate, usually as a consequence of intense coughing, sneezing, or vomiting; a bleeding diatheses; **infectious mononucleosis**; or fellatio. Palatal lesions may also reflect trauma (i.e., physical, chemical, thermal), infection (i.e., bacterial, viral, and fungal), **salivary gland neoplasm**, Kaposi's sarcoma, squamous cell carcinoma, or lymphoma.

By depressing the tongue and asking the patient to say "ah," the oropharynx can be visualized. It is critical to inspect both fauces (tonsillar areas), the anterior pillars (glossopalatine arches), and the posterior pillars (pharyngopalatine arches). These areas are replete with lymphoid tissue and any abnormality should be documented and investigated.

Infectious Mononucleosis

Infectious mononucleosis (IM) is an acute viral illness associated with mild respiratory symptoms and prolonged malaise and fatigue.

The Epstein-Barr virus (EBV) is the main causative agent of infectious mononucleosis. EBV is a human herpesvirus that is present in over 90% of the population. Most exposures occur during adolescence, and the risk of developing IM increases with age of exposure. EBV has also been linked to the development of several malignant tumors, including B-cell neoplasms such as Burkitt's lymphoma and Hodgkin's disease, certain forms of T-cell lymphoma, and undifferentiated nasopharyngeal carcinoma.

Clinical Features

Most EBV infections in infants and children are asymptomatic. However, infection in adolescents or adults often results in symptomatic IM. Classic clinical features include fever, pharyngitis, and lymphadenopathy (Figure 5.55a). Petechiae affecting the soft palate may be present (Figure 5.55b). IM usually resolves over a period of weeks or months without complications.

Diagnosis

The diagnosis of IM is attained by correlating the characteristic clinical and laboratory findings. The most diagnostic laboratory test is the serologic test for heterophil antibodies. Differential diagnosis should include group A β-hemolytic streptococcal pharyngitis or other viral infections.

Treatment

Treatment of uncomplicated IM is supportive and should include nutritional supplementation, hydration, and rest. Acetaminophen may be prescribed for pain and to reduce fever. Aspirin should be avoided, since it has been associated with rare cases of Reye's syndrome during acute EBV infection.

a



b

Figures 5.55a and b. Infectious mononucleosis.

Minor Salivary Gland Neoplasm

Minor salivary gland neoplasms constitute a heterogeneous group of both benign and malignant tumors arising in the oral cavity. The most common benign minor salivary gland neoplasms are the pleomorphic adenoma and the canalicular adenoma. The most common malignant minor salivary gland neoplasms are mucoepidermoid carcinoma, adenoid cystic carcinoma, and the polymorphous low-grade adenocarcinoma. Collectively, minor salivary neoplasms account for less than one-quarter of all salivary neoplasms.

Clinical Features

Minor salivary gland neoplasms typically present as expansile well-defined submucosal nodules or tumors (Figure 5.56 and 5.57). The most common site of occurrence is the palate, but these neoplasms may arise in any area containing minor salivary gland tissue. Advanced lesions may exhibit surface ulceration and local fixation increases the concern of malignancy.

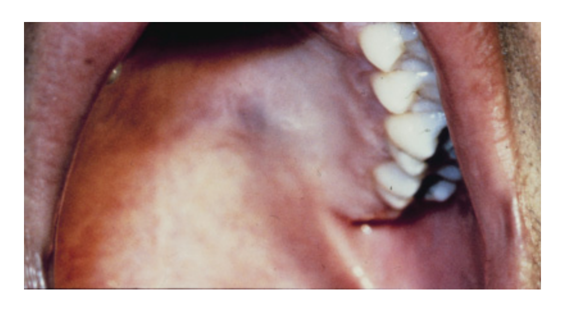


Figure 5.56. Mucoepidermoid carcinoma.



Figure 5.57. Adenoid cystic carcinoma.

Diagnosis

A biopsy is required to diagnose a minor salivary neoplasm. Small lesions should be excised at the time of biopsy.

Treatment

Surgical removal remains the treatment of choice. The prognosis for a benign minor salivary neoplasm is excellent. For malignant salivary gland lesions, the prognosis is greatly influenced by the tumor type and stage at the time of surgery.

Examine the Tongue

The color, size, and shape of the tongue should be assessed and any variations from normal documented. The dorsal surface of the tongue is characterized by the presence of specialized papillae, which can be divided into four groups: filiform, fungiform, circumvallate, and foliate. A history of irregular and shifting areas of shortened filiform papillae, giving the tongue a "geographic" appearance, is characteristic of **erythema migrans** (stomatitis migrans). Excessive lengthening of the filiform papillae on the dorsal tongue is known as **hairy tongue**. The papillae may absorb exogenous stains or harbor chromogenic organisms, often causing a white, yellow, brown, or black discoloration. An atrophic bald erythematous patch affecting the posterior dorsal of the tongue is characteristic of median rhomboid glossitis (see **"Candidiasis"**). Numerous **nutritional deficiencies** may result in the tongue becoming beefy red, ulcerated or pale, and atrophic. A tongue that appears edematous and cyanotic may be observed in

polycythemia vera, a condition of excess bone marrow activity.

Grasping the tongue with dry gauze and gently pulling it from side to side allows the clinician to inspect the lateral, ventral, and posterior aspects of the tongue. Scalloping or indentations along the lateral border may indicate the presence of a tongue sucking habit, a clenching habit, or macroglossia. Common causes of macroglossia include normal variation, vascular lesions (e.g., hemangioma, lymphangioma), acromegaly, and myxedema (hypothyroidism).

Traumatic ulcers, papillomas, and fibromas frequently occur on the tongue. The tongue is the most common location for a **granular cell tumor** and **hairy leukoplakia**. The ventral surface of the tongue often reveals pronounced varicosities, usually as a function of age. The incidence of squamous cell carcinoma of the tongue is second only to squamous cell carcinoma of the lips. Because of the tendency for carcinoma of the tongue to undergo early metastasis, the clinician must evaluate all erythroplakic, leukoplakic, and ulcerative lesions of the tongue with a high degree of suspicion.

Erythema Migrans

Erythema migrans is a poorly understood benign condition characterized by focal areas of erythema or atrophy. Putative associations with several conditions such as psoriasis, allergies, diabetes mellitus, hormonal disturbances, nutritional disturbances, psychological disorders, lichen planus, Down syndrome, and Reiter's syndrome remain largely unproven. Erythema migrans affects approximately 1–2.5% of the population.

Clinical Features

Erythema migrans typically presents as multiple circinate or irregular erythematous patches, which are marginated by whitish to yellowish keratotic lines (Figures 5.58, 5.59, 5.60a, and 5.60b). By far the most common site of occurrence is the dorsal tongue, in which case the erythematous patches correspond to a loss of the filiform papillae. Pain is rarely observed, but some patients complain of burning or itching of the affected sites.

Diagnosis

The characteristic appearance is pathognomonic and biopsy should be reserved for equivocal cases. The differential diagnosis should include the putatively associated conditions mentioned above.

Treatment

Simple recognition and patient reassurance will usually suffice. For patients with symptomatic erythema migrans, a topical antihistamine solution, such as diphenhydramine

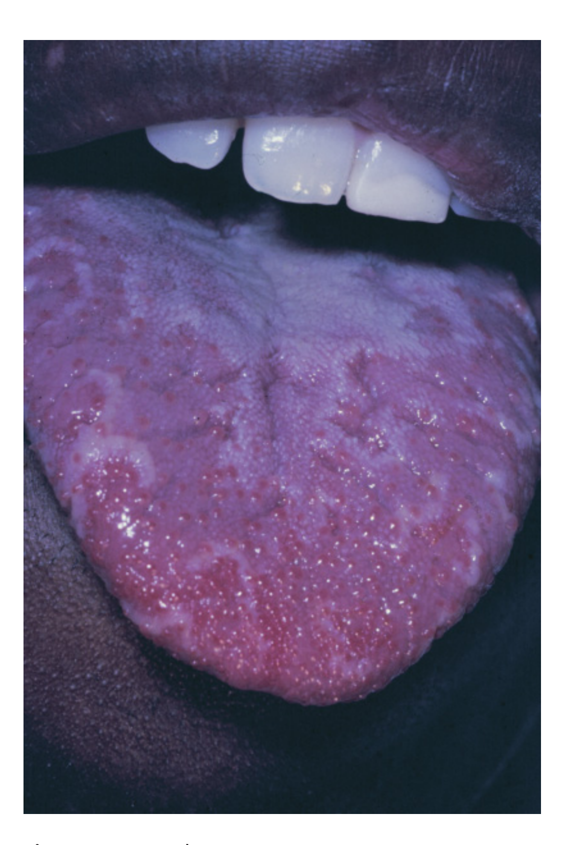


Figure 5.58. Erythema migrans.



Figure 5.59. Erythema migrans.



a



b

Figures 5.60a and b. Erythema migrans.

elixir, or a steroid rinse may be prescribed to reduce symptoms.

Hairy Tongue

Hairy tongue (HT) is a commonly observed benign condition associated with reduced desquamation or excess formation of the filiform papillae. Normal filiform papillae are approximately 1 mm in length, whereas filiform papillae in HT may exceed 1.5 mm in length. The specific underlying cause of HT is unclear. Predisposing factors include poor oral hygiene, smoking, alcohol, mouthwash use, numerous medications, and therapeutic radiation to the head and the neck. The prevalence varies widely among population groups, but overall it is quite common.

Clinical Features

HT is characterized by a hypertrophy and elongation of the filiform papillae. The most frequently observed form is referred to as black hairy tongue; however, HT may also appear brown, white, green, or any of a variety of hues (Figures 5.61, 5.62, and 5.63). It is rarely symptomatic, but interspersed deepened furrows may harbor candidal overgrowth. Patients may also develop halitosis as a result of retention of oral debris between the elongated papillae.

Diagnosis

The diagnosis of HT is straightforward and is based principally on the clinical presentation. The perception of halitosis may prompt

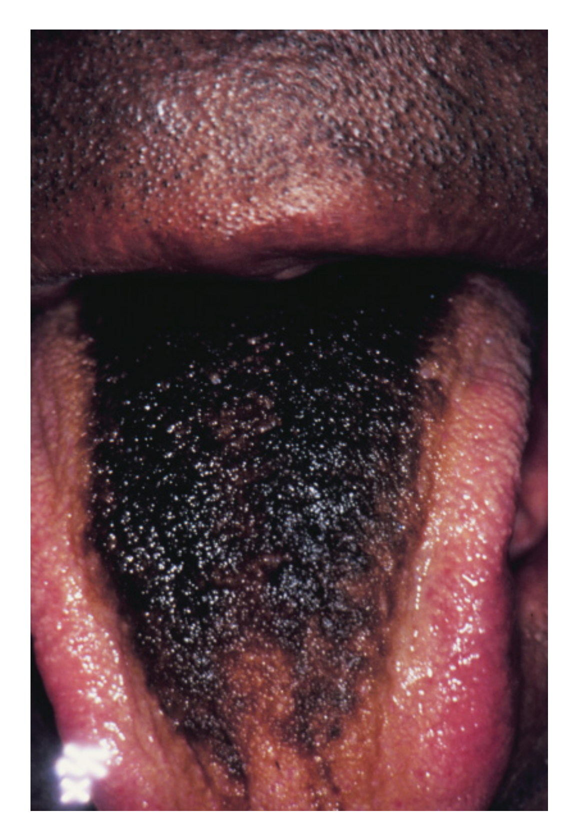




Figure 5.61. Hairy tongue.

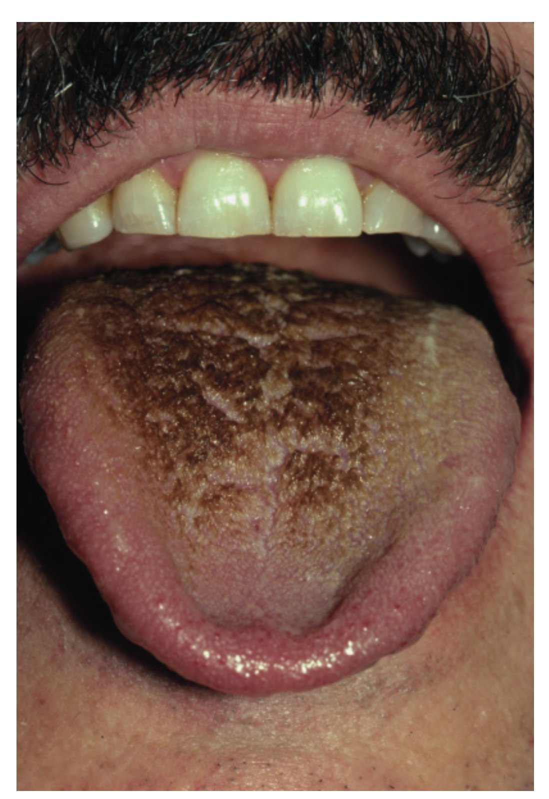


Figure 5.62. Hairy tongue.

the patient to report to the dental office for an evaluation.

Treatment

Management of HT consists of removing known predisposing factors and improving oral hygiene. Simple tongue brushing is often sufficient to reduce HT. In rare cases, physical removal of the elongated filiform papilla may be accomplished with electrodessication, carbon dioxide laser, or even clipping. If a candidal infection is present, topical antifungal medications should be prescribed.



Figure 5.63. Hairy tongue.

Nutritional Deficiencies

Numerous nutritional deficiencies may induce a variety of changes affecting the oral mucosa. Commonly implicated nutrients include B vitamins (B_{12}, niacin, riboflavin, pyridoxine), iron, folic acid, and vitamin C. Underlying conditions associated with inadequate or impaired absorption, such as alcoholism, malabsorptive disorders, and eating disorders, further add to the risk.

Clinical Features

The oral presentation of a patient suffering from a nutritional deficiency is variable and rarely specific. The tongue is the most frequently affected oral site. Two common descriptive entities are "beefy red tongue" and "pale tongue with papillary atrophy." The presence of pallor is suggestive of iron deficiency (Figures 5.64 and 5.65), while beefy red tongue is more typically associated with vitamin B_{12} or folic acid deficiency (Figure 5.66a). The concomitant

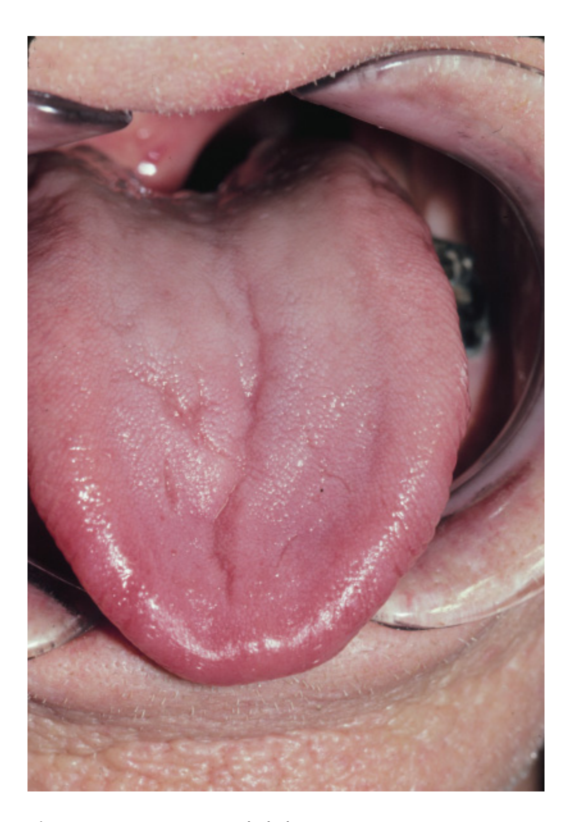


Figure 5.64. Nutritional deficiency.



Figure 5.65. Nutritional deficiency.

a 

 b

Figures 5.66a and b. Nutritional deficiency.

occurrence of angular cheilitis with either of these conditions is frequently observed (Figure 5.66b). The patient may also complain of a burning or tingling pain.

Diagnosis

The diagnosis of a nutritional deficiency is established via appropriate laboratory tests.

Treatment

Medical therapy to address any underlying systemic factors, combined with necessary nutritional supplementation, is usually sufficient to correct the deficiency.

Granular Cell Tumor

The granular cell tumor (GCT) is an uncommon neoplasm believed to arise from either the Schwann cell or an undifferentiated mesenchymal cell. Women are affected more often than men (3 : 1), and most tumors are discovered between the fourth and sixth decades.

Clinical Features

Most GCTs occur in the head and neck region, and there is a predilection for the tongue. A GCT typically presents as a solitary, slow-growing, painless, nonulcerated, submucosal nodule or tumor (Figures 5.67 and 5.68). The surface mucosa is either normal or slightly pinkish in color.

Diagnosis

A biopsy is required to confirm the diagnosis. Other lesions to consider in a differential diagnosis include a fibroma, lipoma, neural tumor, hemangioma, and lymphangioma. The GCT may exhibit histological evidence of pseudoepitheliomatous hyperplasia, and thus should be evaluated by an experienced pathologist.

Treatment

Surgical excision is the treatment of choice, and recurrence is rare.

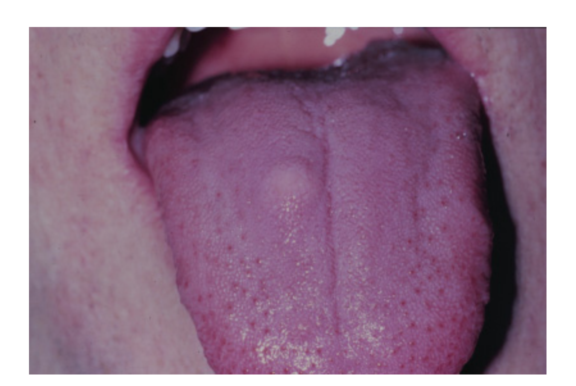


Figure 5.67. Granular cell tumor.



Figure 5.68. Granular cell tumor.

Hairy Leukoplakia

Hairy leukoplakia (HL) is a unique type of leukoplakia that is almost always associated with immunosuppression. While the exact pathogenic mechanism remains to be determined, it is accepted that the presence of EBV is necessary for the development of HL.

Clinical Features

HL almost always affects the lateral and dorsolateral aspects of the tongue (Figures 5.69a and 5.69b). It presents as multiple white vertical folds or corrugations that cannot be wiped off. Lesions vary in size from almost imperceptible to large plaques that may interfere with normal eating and speaking.

Diagnosis

The rather characteristic clinical appearance, combined with a history of immunosuppression, allows for a straightforward clinical diagnosis. A biopsy is rarely necessary. If performed, immunohistochemical staining for EBV should be used to confirm the diagnosis.

Treatment

HL is generally asymptomatic and rarely requires treatment. HL will generally regress following treatment of the underlying cause of immunosuppression.

Examine the Glossopharyngeal (IX) and Vagus (X) Nerves

The glossopharyngeal nerve provides sensory function to the posterior one-third of the tongue, soft palate, and pharynx; special sense of taste to the posterior one-third of the tongue and soft palate; motor function to the stylopharyngeus muscle; secretory and motor function to the parotid glands; and it initiates the gag reflex. The vagus nerve provides overlapping motor function with the glossopharyngeal nerve for the gag reflex; sole motor innervation to the laryngeal muscles (voice); sensory innervation to the pinna of the ear; and parasympathetic innervation to the thoracic and abdominal viscera. Vagal nerve paralysis, possibly caused by diphtheria, may result in the patient having a nasal voice and food regurgitation through the nose. Clinically, the uvula may be deflected to the unaffected side.

a 

 b

Figures 5.69a and b. Hairy leukoplakia.

Assess Sensory and Motor Function

The gag reflex varies among patients, but can usually be stimulated and assessed by gently stroking the pharyngeal tissue with an applicator stick. Likewise, stroking the left and right side of the uvula is used to test the palatal reflex, whereupon the stroked side should rise. When the patient is instructed to say "ah," the entire soft palate should rise.

The hypoglossal nerve provides motor function to the tongue. Injury to the nerve, often as a consequence of surgery or trauma, results in paralysis of the tongue on the affected side. When the tongue is protruded, it will deviate to the affected side and, in time, show evidence of muscle atrophy. Additional signs of hypoglossal nerve damage are slurred speech, difficulty masticating, and an inability of the patient to resist contralateral pressure applied to the tongue.

Examine the Floor of the Mouth

The mylohyoid muscles support the floor of the mouth. Inspection and bimanual palpation of the area permit the identification of lesions arising within the supporting tissues of the region, in the major salivary glands and their ducts, and in the submandibular and submental lymph nodes. Anatomically, the sublingual glands and the superior portion of the submandibular glands lie superior to the mylohyoid muscles. The sublingual glands and the superior lobes of the submandibular glands are palpable orally. Salivary flow may be assessed by blotting dry the area of the lingual caruncle (Wharton's duct). Pressure may be applied to the submandibular area to express saliva, which will usually pool in the floor of the mouth. Anticholinergic drugs, sialoliths, infection, Sjögren's syndrome, or neoplasia may restrict salivary flow. A **ranula** is a variant of a mucocele affecting the sublingual or submandibular glands. Squamous cell carcinoma of the floor of the mouth typically appears as an area of **erythroplakia**, **leukoplakia**, and/or a painless ulceration with rolled indurated borders.

Ranula

The ranula is a relatively uncommon phenomenon caused by blockage of the sublingual or submandibular glands. It is generally accepted that most cases result from extravasation of saliva into the connective tissue adjacent to the sublingual gland. The prevalence of a ranula is unknown, but it appears to be more frequently observed in individuals infected with HIV.

Clinical Features

The ranula usually presents as a blue-domed, translucent swelling in the floor of the mouth, resembling the "belly of a frog" (Figure 5.70). If the ranula extends down through the mylohyoid muscle, it is called a plunging ranula. This lesion manifests as a painless, nonmobile swelling in the neck.



Figure 5.70. Ranula.



Figure 5.71. Erythroplakia.

Diagnosis

A classic ranula is easily recognized; however, the plunging ranula may occur without any concurrent intraoral involvement, necessitating further assessment for confirmation. Advanced imaging techniques, particularly magnetic resonance imaging (MRI), combined with fine needle aspiration may be useful to assess these lesions. Other lesions to consider in the differential diagnosis include a hemangioma, lymphangioma, and salivary gland neoplasm.



Figure 5.72. Erythroplakia.

Treatment

Treatment options for the ranula include excision of the lesion with or without excision of the ipsilateral sublingual or submandibular gland, marsupialization, cryosurgery, and CO_2 laser excision. The choice of treatment is dependent upon the location and the size of the ranula, and the skill and experience of the surgeon.

Erythroplakia

Erythroplakia is a descriptive clinical term for any red macular lesion affecting the oral mucosa that cannot be given a specific clinical diagnosis. Erythroplakia is usually diagnosed in the sixth to seventh decade, and most patients relate a positive history of long-term tobacco use and alcohol consumption. The prevalence of erythroplakia in the United States is unknown, but estimates of up to 0.83% have been reported in South and Southeast Asia. Histological assessment of erythroplakia reveals the presence of dysplasia, carcinoma-in-situ, or invasive squamous cell carcinoma in over 90% of cases.

Clinical Features

Erythroplakia may manifest as a homogenous red macule, a mixed macular red-and-white lesion, or as a red lesion with superimposed white granular spots (speckled leukoplakia) (Figures 5.71, 5.72, 5.73, and

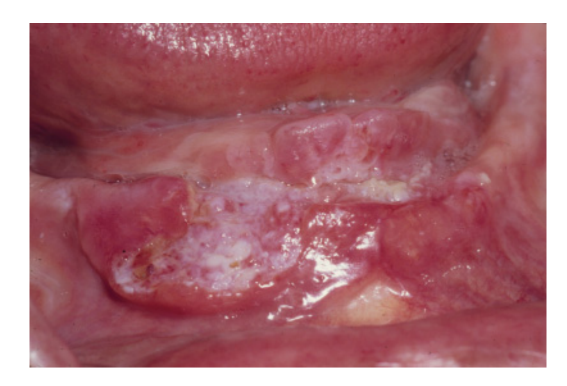


Figure 5.73. Erythroplakia.



Figure 5.74. Erythroplakia.

5.74). Lesions are most prevalent in the ventral and lateral aspects of the tongue, the retromolar-trigone-soft palate complex, the floor of the mouth, and buccal mucosa. Although often asymptomatic, some patients may complain of discomfort, especially when eating hot or spicy food.

Diagnosis

Erythroplakia is strictly a clinical diagnosis, and it infers a high degree of suspicion for malignancy. Any suspicious lesion that persists for 2 weeks should be biopsied. The importance of recognizing and evaluating any persistent (over 2 weeks) erythroplakia cannot be overemphasized, as evidenced by the fact that dysplasia, carcinoma-in-situ, or invasive SCC is diagnosed microscopically in well over 90% of the lesions characterized clinically as erythroplakia.

New products marketed to assist a clinician's assessment in identifying areas of highest suspicion, such as Oral CDx®, ViziLite®, ViziLite® Plus, and VELscope®, do not obviate the need to perform a biopsy.

Treatment

Ablative therapies used to treat erythroplakia include surgical excision, eletrocoagulation, cryosurgery, and laser surgery. The choice of therapy is based upon clinician experience and patient desires. Counseling about tobacco and alcohol use and close long-term monitoring are indicated.

Leukoplakia

Leukoplakia is a descriptive term for a white lesion of the oral mucosa that cannot be attributed to any other clinically definable lesion. The prevalence of leukoplakia in the United States is unknown, but it is estimated to approach 3%. The rate of malignant transformation of leukoplakia cannot be predicted accurately, but it is important to acknowledge that up to 85% of all precancerous lesions are leukoplakic.

Clinical Features

Leukoplakia may present as a homogeneous white lesion or as a nonhomogenous speckled, ulcerated, or papillary white-and-red



Figure 5.75. Leukoplakia.

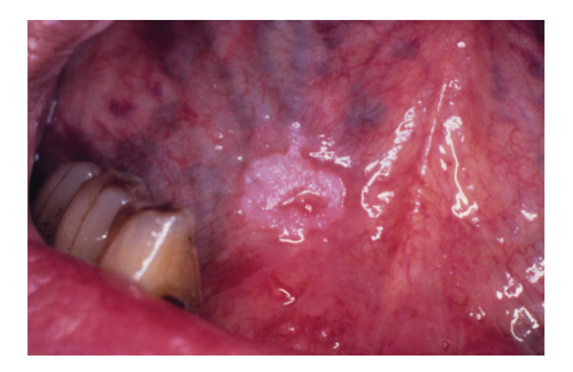


Figure 5.76. Leukoplakia.

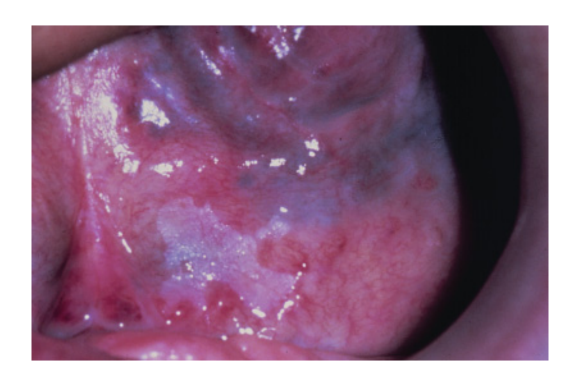


Figure 5.77. Leukoplakia.

lesion (Figures 5.75, 5.76, 5.77, and 5.78). It is most prevalent in the buccal mucosa and mandibular sulcular/alveolar ridge areas, the floor of the mouth, the ventral and lateral aspects of the tongue, the palate, and gingivae.

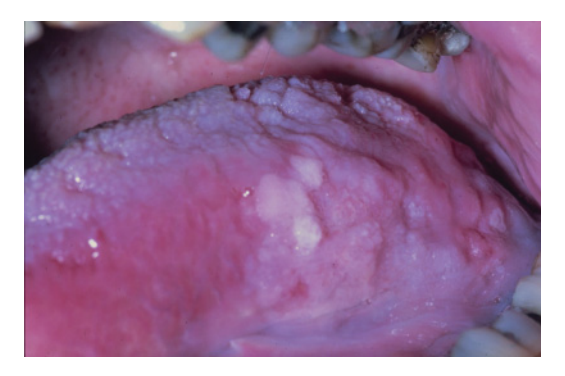


Figure 5.78. Leukoplakia.

Diagnosis

Similar to erythroplakia, leukoplakia is strictly a clinical term. As a rule, any lesion that persists beyond 2 weeks should be biopsied. Conditions to consider in a differential diagnosis include frictional keratosis, factitial injury, lichen planus, lupus erythematosus, white sponge nevus, leukoedema, and hyperplastic candidiasis.

Treatment

Ablative therapies used to treat leukoplakia include surgical excision, eletrocoagulation, cryosurgery, and laser surgery. The choice of therapy is based upon clinician experience and patient desires. Counseling about tobacco and alcohol use and close long-term monitoring are indicated.

Examine the Gingivae

The gingiva is a keratinized band of tissue that surrounds and attaches to the root surface of each tooth. The attachment extends onto the periosteum of the supporting alveolar bone. Visual and tactile examination allows for an assessment of gingival color, consistency, texture, architecture, and relationship to adjacent structures. Gingival pigmentation is most common in dark-

skinned individuals and is usually physiologic. Discolored or dark marginal gingivae may occur as a consequence of excess exposure to lead, mercury, and bismuth. Gingival inflammation or gingivitis is characterized by gingival erythema and edema, and is usually due to local factors such as poor oral hygiene and plaque accumulation. However, gingivitis may be associated with numerous systemic conditions.

Necrotizing ulcerative gingivitis is a painful inflammation of the gingivae with an unmistakable fetid odor. **Gingival hyperplasia** presents as enlarged, variably inflamed, firm, or edematous gingivae. Desquamative gingivitis is a descriptive term used to describe sloughing gingival tissues and may be the early presenting sign of a mucocutaneous disease (e.g., lichen planus, pemphigus, and **mucous membrane pemphigoid**). A common clinical presentation of **herpetic infections** is primary herpetic gingivostomatitis.

Spontaneous gingival bleeding may be seen in association with gingivitis, thrombocytopenia, polycythemia vera, aplastic anemia, hemophilia, and drug therapy (e.g., antithrombotic agents, warfarin, and heparin). Some of the more common exophytic lesions that occur on the gingivae are a **pyogenic granuloma**, **peripheral giant cell granuloma**, and **peripheral ossifying fibroma**. Less frequently occurring lesions include carcinomas and sarcomas.

Necrotizing Ulcerative Gingivitis

Necrotizing ulcerative gingivitis (NUG) is a bacterial infection of the gingivae associated with elevated levels of *Prevotella intermedia* and other pathogenic spirochetes. Predisposing factors include poor oral hygiene, smoking, poor nutrition, stress, and immunosuppression. Most cases occur in young to middle-aged adults, and the exact prevalence is unknown.

Clinical Features

The hallmark features of NUG are pain, gingival ulceration with necrosis, and bleeding (Figures 5.79 and 5.80). The interdental papillae frequently demonstrate a "punched out" appearance with loss of vertical height. Other common signs and symptoms include fetid breath, sialorrhea, metallic taste, fever, and lymphadenopathy. Extension of the disease to include the underlying alveolar osseous tissues can lead to necrotizing ulcerative periodontitis (NUP).

Diagnosis

The abrupt onset of the characteristic presentation of NUG makes for an easy clinical diagnosis. Bacterial culturing is not indicated in the routine clinical setting.



Figure 5.79. Necrotizing ulcerative gingivitis.



Figure 5.80. Necrotizing ulcerative gingivitis.

Treatment

The treatment of NUG consists of simple debridement and the institution of improved oral hygiene. Antibiotic therapy may be considered for cases presenting with fever and lymphadenopathy. Nonresponsive cases must be evaluated further to rule out the presence of more serious systemic conditions such as immunosuppression or leukemia.

Gingival Hyperplasia

Gingival hyperplasia is a clinical diagnosis characterized by gingival enlargement. Potential causes of gingival hyperplasia include inflammation, hereditary predisposition, certain syndromes, and drug exposure. The most frequently implicated medications are the anticonvulsant drugs phenytoin and valproic acid, the immunosuppressant drug cyclosporine, and numerous calcium channel blocking agents. The prevalence of gingival hyperplasia in the normal population is estimated to vary between 4.0% and 7.5%.

Clinical Features

Gingival hyperplasia initially manifests as generalized enlargement involving the interproximal papillae (Figures 5.81, 5.82, 5.83, and 5.84). Progression results in more widespread involvement and extension of the gingival tissues around the teeth. Severe cases may eventually spread to cover the clinical crowns of the dentition. In many cases, the edentulous areas will be spared.



Figure 5.81. Gingival hyperplasia.



Figure 5.82. Gingival hyperplasia.



Figure 5.83. Gingival hyperplasia.



Figure 5.84. Gingival hyperplasia.

Diagnosis

The diagnosis is straightforward. A thorough medical history is necessary to identify any contributing factors such as medication exposure or genetic predisposition.

Treatment

Drug-induced gingival hyperplasia may improve if the offending drug can be identified and discontinued. However, most cases of gingival hyperplasia require surgical therapy to remove redundant tissue. If contributing factors are not removed, recurrence is likely.

Mucous Membrane Pemphigoid

Mucous membrane pemphigoid (MMP), a form of autoimmune vesiculoulcerative disease, has a predilection for involving the oral and conjunctival mucosa. Affected patients develop autoantibodies that target the basement membrane zone of the epithelium. The typical patient is a woman between the ages of 60 and 80 years.

Clinical Features

The onset of MMP is insidious, and the most commonly affected oral site is the gingivae (Figures 5.85a–5.85d). The gingivae often demonstrate erythema and desquamation. Signs of ocular involvement include conjunctivitis, burning, and photophobia.

Diagnosis

A biopsy and immunofluorescent studies are required to establish a definitive diagnosis. Other conditions to consider in a differential diagnosis include pemphigus vulgaris, erythema multiforme, herpetic simplex infection, lichen planus, and lupus erythematosus.

Treatment

There is no cure for MMP. Topical or systemic immunosuppressive agents may be



Figures 5.85a–d. Mucous membrane pemphigoid.

useful to manage oral lesions. Systemic therapies should be accomplished in close consultation with the patient's physician. All MMP patients should receive an ophthalmologic examination to rule out ocular involvement.

Herpetic Infections

The herpes simplex virus (HSV) is a ubiquitous virus that is spread through contact with infected secretions. Two strains are acknowledged: HSV-1, which is associated with oral infections, and HSV-2, which is associated with genital infections. However, this site specificity is not absolute. Most cases of HSV-1 occur in children. Exposure rates as high as 90% have been reported, and age-specific prevalence rates appear to be decreasing in industrialized countries.

A unique feature of all herpes viruses is their ability to establish latency in an infected host. The most frequent site of latency for HSV-1 is the trigeminal ganglion. Reactivation of the latent virus may occur following ultraviolet light exposure, mechanical trauma, fever, immunosuppression, decompression of the trigeminal nerve, and dietary factors. About 40% of patients with prior exposure experience recurrent infection, and approximately 100 million cases of recurrent infections occur each year in the United States.

Clinical Features

Two stages of infection are recognized, primary and recurrent. In reality, over 90% of primary infections result in either asymptomatic or mildly symptomatic illness. The classical clinical illness is primary herpetic gingivostomatitis. After a prodromal period characterized by malaise, irritability, headache, and fever, patients develop gingivitis, but painful vesicular eruptions may arise on any oral mucosal surface (Figures 5.86a–5.86d). Within 24–48 hours, the vesicles rupture producing small, round, shallow,



Figures 5.86a–d. Primary herpetic gingivostomatitis.

and painful erosions. Vesicles and erosions often coalesce to form large irregular lesions that heal within 7–14 days. Associated pain may be so severe as to adversely affect the patient's ability to eat, swallow, and speak.

Over 90% of recurrent herpetic infections occur on the lips (recurrent herpes labialis) unaccompanied by systemic illness (Figures 5.87a–5.87d). The patient may relate a prodromal period of hyperesthesia or altered sensation, erythema, and edema at the site of involvement. Within hours, a localized vesicular eruption develops, which subsequently coalesces and crusts over during the next few days. Total resolution is noted in about 10–14 days.

Recurrent herpetic infections may also occur intraorally, where they tend to affect the keratinized mucosa of the hard palate or gingivae (Figures 5.88, 5.89, 5.90, and 5.91).

Figures 5.87a–d. Recurrent herpes labialis.

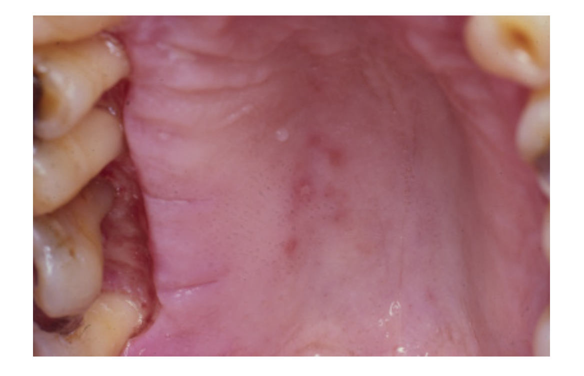


Figure 5.88. Recurrent intraoral herpes.



Figure 5.89. Recurrent intraoral herpes.

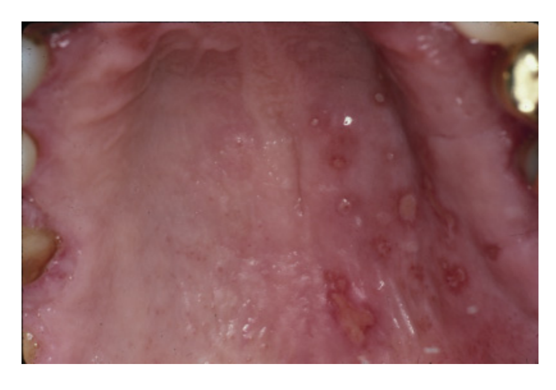


Figure 5.90. Recurrent intraoral herpes.



Figure 5.91. Recurrent intraoral herpes.

While most cases of primary and recurrent HSV infections resolve uneventfully, immunocompromised patients are at risk for developing severe complications, for example, encephalitis and blindness.

Diagnosis

The diagnosis of primary or recurrent HSV-1 infections is typically made based upon history and the characteristic clinical presentation. Specific laboratory tests are available but are rarely necessary to establish a diagnosis. Conditions to consider in a differential diagnosis include herpetiform recurrent aphthous stomatitis, herpangina, pemphigus, erythema multiforme, necrotizing ulcerative gingivitis, and hand-foot-and-mouth disease.

Treatment

For primary infections, the goal of therapy is to provide palliative and supportive care. Successful management consists of controlling fever and pain, preventing dehydration, shortening the duration of lesions, and monitoring for systemic viremia. Recurrent herpes labialis often does not require treatment. Several over-the-counter and prescription products are marketed to relieve pain and promote healing. The use of lip balms and lotions with an SPF of at least 15 may prevent sun-induced recurrent infections. In all cases, proper hygiene to reduce the risk of transmission and autoinoculation should be enforced. Antiviral chemotherapy should be considered for patients at an increased risk of developing systemic dissemination.

Pyogenic Granuloma

The pyogenic granuloma is a commonly observed reactive inflammatory response to local irritation. The exact etiology is often unknown but most cases occur as a response to an irritant such as plaque, calculus, or foreign material. Hormonal alterations likely modulate the development of a pyogenic granuloma, as evidenced by a distinct female predilection and an increased risk of occurrence during pregnancy (pregnancy tumor). The exact prevalence is unknown.

Clinical Features

The pyogenic granuloma presents as a bright red pedunculated or sessile mass of variable size (Figures 5.92 and 5.93). These lesions are highly vascular, bleed easily, and often exhibit exuberant growth. The most common location is the gingivae.

Diagnosis

While the clinical appearance is highly suggestive, particularly during pregnancy, a biopsy is recommended for persistent lesions.



Figure 5.92. Pyogenic granuloma.



Figure 5.93. Pyogenic granuloma.

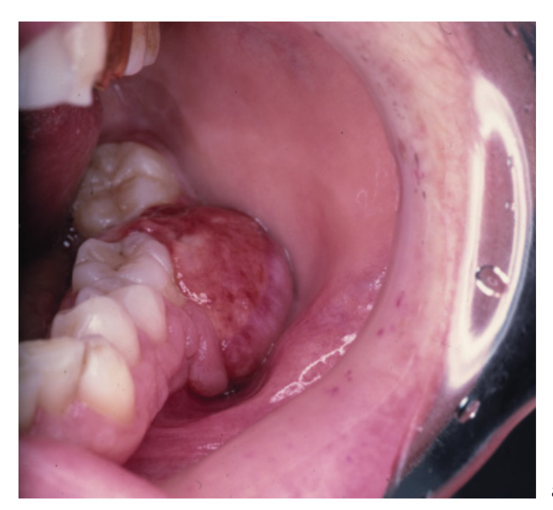
A differential diagnosis should include a fibroma, peripheral ossifying fibroma, the peripheral giant cell granuloma, and a benign neural tumor.

Treatment

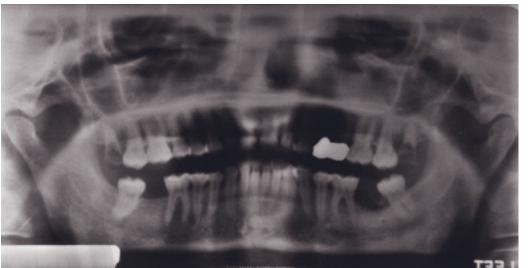
A small pyogenic granuloma may undergo spontaneous involution after the implementation of improved oral hygiene or after delivery. Persistent or large lesions should be excised and the adjacent teeth should be thoroughly scaled and curetted.

Peripheral Giant Cell Granuloma

The peripheral giant cell granuloma (PGCG) is best considered as the soft-tissue counter-



a



b

Figures 5.94a and b. Peripheral giant cell granuloma.

part of the central giant cell granuloma. The etiology and prevalence are unknown. Most lesions occur in young adult females, and there is a predilection for the mandible.

Clinical Features

A PGCG is clinically indistinguishable from a pyogenic granuloma. Compared with the pyogenic granuloma, the color may appear more deeply purple, and the PGCG only arises on gingivae or edentulous alveolar ridge (Figure 5.94a). Superficial osseous erosion or cupping of the underlying bone may be noted on a radiograph (Figure 5.94b).

Diagnosis

A biopsy is required to establish a definitive diagnosis. Conditions that may mimic the

PGCG include a fibroma, a pyogenic granuloma, peripheral ossifying fibroma, and benign neural tumor.

Treatment

Surgical excision is the treatment of choice, followed by thorough scaling and curettage of the affected teeth.

Peripheral Ossifying Fibroma

The peripheral ossifying fibroma (POF) is a reactive hyperplastic inflammatory lesion. It is considered to arise from the periosteum or from periodontal ligament mesenchymal cells in response to local irritants such as plaque, calculus, and foreign material. Females are affected more often than males, and most patients are between the ages of 5 and 25.

Clinical Features

A POF usually presents as a well-circumscribed nodular mass usually arising from the anterior gingival mucosa (Figure 5.95). The surface may be smooth or ulcerated. Like the PCGC, POF only arises on the gingiva or edentulous alveolar ridge. POF frequently causes separation and/or divergence of the adjacent teeth (Figures 5.96a and 5.96b). Occasionally, it may be associated with minimal underlying osseous resorption.

Diagnosis

A biopsy is necessary in order to establish a definitive diagnosis. Other conditions to consider in a differential diagnosis include a pyogenic granuloma, peripheral giant cell granuloma, fibroma, or an odontogenic tumor.

Treatment

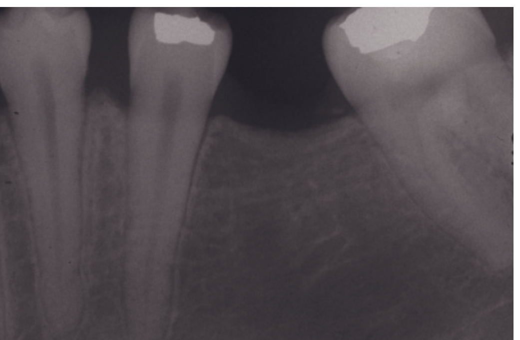
Treatment consists of surgical excision down to the periosteum and the elimination of any local irritants. Tooth extraction is seldom necessary, and the prognosis is quite good. Recurrence rates as high as 16% have been reported.



Figure 5.95. Peripheral ossifying fibroma.



a



b

Figures 5.96a and b. Peripheral ossifying fibroma.

Examine the Teeth

Note the Number of Teeth

Developmental disturbances affecting the number of teeth may manifest as partial or total anodontia or hypodontia and supernumerary teeth (hyperdontia). Hypodontia is a relatively common finding in the permanent dentition, and the most common missing teeth are third molars, mandibular second premolars, maxillary second premolars, and maxillary lateral incisors, respectively. If several teeth are missing, the possibility of ectodermal dysplasia, Reiter's syndrome, or incontinentia pigmenti should be considered. Hyperdontia or supernumerary teeth are commonly found in the anterior maxilla as mesiodens and in the molar area, usually as fourth molars. Hyperdontia may also be the manifestation of cleidocranial dysplasia and Gardner's syndrome.

Note the Size of Teeth

Developmental disturbances affecting the size of teeth may manifest as microdontia, macrodontia, and taurodontism. Microdontia is characterized by smaller than normal teeth and may be secondary to pituitary hypofunction during childhood. Macrodontia is characterized by larger than normal teeth and may be secondary to pituitary hyperfunction during childhood. Taurodontism is characterized by radiographic evidence of abnormally large pulp chambers extending apically.

Note the Shape of Teeth

Developmental disturbances affecting the shape of teeth may manifest as germination, fusion, concrescence, and dens invaginatus. Germination usually affects permanent incisors and is believed to be the result of a tooth germ attempting to divide, resulting in a double crown or a double root. Fusion, more common in the primary dentition, is the result of two adjacent tooth germs joined by enamel and/or dentin. Concrescence is characterized by radiographic evidence of the roots of two adjacent teeth joined by cementum. Dens invaginatus commonly affects maxillary lateral incisors and manifests as invagination (sometimes into the pulp) on the palatal surfaces of affected teeth.

Note the Color of Teeth

Inherited disturbances affecting the color of teeth include **amelogenesis imperfecta** and dentinogenesis imperfecta. In amelogenesis imperfecta, the enamel is often absent and the exposed dentin has a yellowish to brown discoloration. The color of the teeth in dentinogenesis imperfecta and osteogenesis imperfecta is opalescent, ranging from a slightly golden brown to a bluish tinge.

Reactive disturbances affecting the color of teeth may be secondary to inflammation, vitamin deficiencies, trauma, and exposure to tetracycline or excess fluoride. An isolated tooth with a grayish-blue hue may indicate the presence of a necrotic pulp, often due to prior trauma. A tooth that appears pink (pink tooth of Mummery) invariably signals the presence of internal resorption. Yellow to violet staining that fluoresces under ultraviolet light characterizes tetracycline staining. **Fluorosis** or "mottled enamel" is caused by ingestion of fluoride in the drinking water in excess of 1.5 parts per million. The extent of discoloration may vary from mild chalky white blotching to yellow and even brown staining and is dependent on the extent of fluoride exposure.

Amelogenesis Imperfecta

Amelogenesis imperfecta (AI) is a heterogeneous inherited disorder with defective tooth enamel formation caused by various gene mutations in the absence of any generalized or systemic disease. AI has an estimated

prevalence of 1 in 718 to 1 in 14,000, depending on the population studied.

Clinical Features

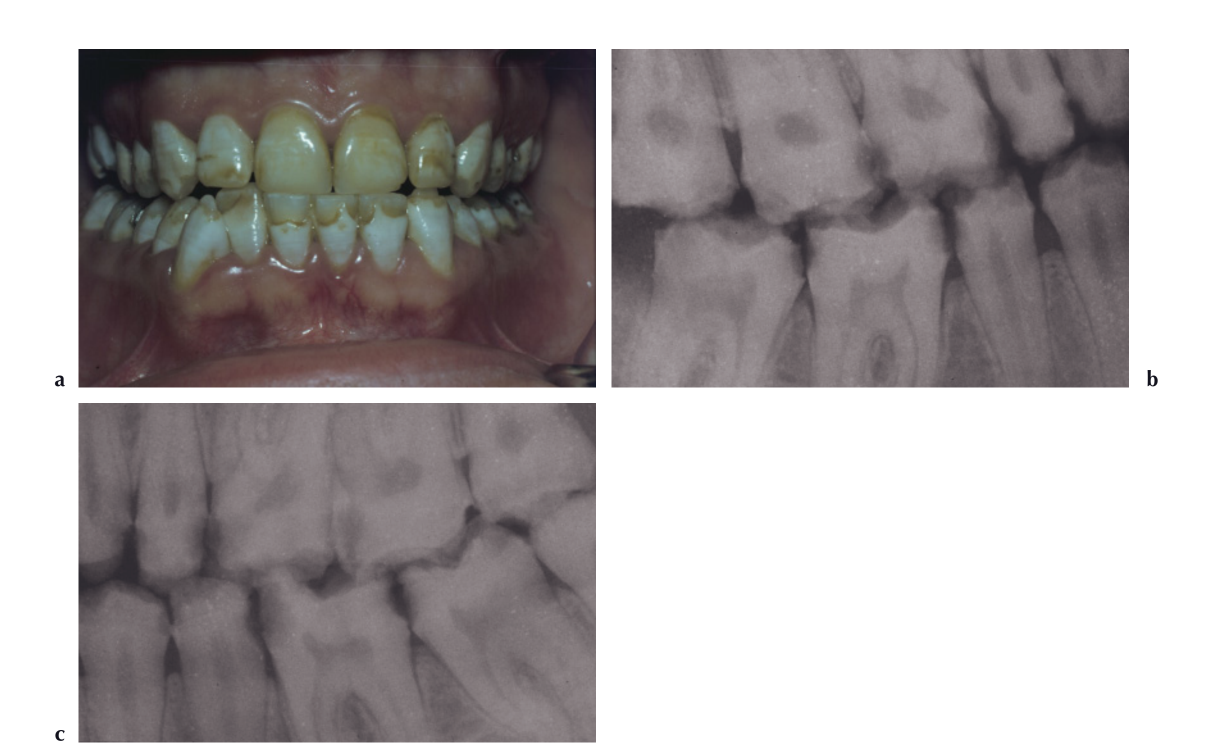
The clinical presentation of AI may include a reduction in the amount of enamel produced (hypoplasia) and/or a defect in mineralization of the enamel (hypomineralization). The teeth may have fine enamel or grooves and pits scattered across the surface of the teeth arranged in rows or columns; exhibit a mottled, opaque white-brown yellow discoloration; or show enamel that has a very low mineralization, manifested clinically by pigmented, softened, and easily detachable enamel (Figures 5.97a–5.97c).

Diagnosis

The diagnosis of defects in dental enamel must be based on clinical, radiographic, and, when possible, laboratory data. Although molecular and biochemical methods have shown differences in the composition of the enamel with some types of AI, other AI-associated gene defects remain unknown, making routine clinical and radiographic observations extremely important. In the differential diagnosis, the clinician should consider acquired defects (chemical insult, nutritional deficiencies, infections, and trauma), dentinogensis imperfecta, and tricho-dento-osseous syndrome.

Treatment

The management depends on the severity of the problem. Treatment strategies include composite resin veneers and jacket crowns for anterior teeth, as well as steel crowns for posterior teeth. Full crowns will improve cosmetic appearance and protect the teeth from damage.



Figures 5.97a–c. Amelogenesis imperfecta.

Fluorosis

Dental fluorosis (DF) is a permanent hypomineralization of enamel resulting from the ingestion of excessive amounts of fluoride (chronic exposure) during tooth formation. Fluoride only causes fluorosis in concentrations of greater than 1 ppm. The main documented risk factors for fluorosis are use of fluoridated drinking water, fluoride supplements, fluoride toothpaste, and infant formulas reconstituted with fluoridated water used before the age of 6 years. It is now believed that fluorosis occurs when fluoride interacts with mineralizing tissues, causing alterations in the mineralization process.

Clinical Features

Fluorosis varies in appearance from white striations to stained pitting of enamel (Figures 5.98a–5.98c). The very mild and mild forms of enamel fluorosis appear as small, barely visible, white flecks found primarily on cusp tips and on facial surfaces of a tooth's enamel surface that are not readily apparent to the affected person or casual observer. In the moderate form, more than 50% of the enamel surface is opaque white. The rare, severe form manifests as pitted and brittle enamel. After eruption, teeth with moderate or severe fluorosis might develop areas of brown stain. In the severe form, the compromised enamel might break away, resulting in excessive wear of the teeth. Fluorosis of the primary teeth occurs less often and is milder than that of the permanent teeth.

Diagnosis

Diagnosis of DF is based on clinical presentation and history of excessive fluoride intake. Definitive diagnosis requires that the defect is present bilaterally in a symmetric pattern,



Figures 5.98a–c. Fluorosis.

and evidence of prior excessive fluoride intake or elevated levels of fluoride in the enamel or other tissues should be found.

Treatment

Preventive management of dental fluorosis includes de-fluoridation of drinking water in endemic areas, water low in fluoride for dilution of infant formulas, cautious use of fluoride supplements and supervision of the use of fluoride toothpaste by children aged below 5 years. Aesthetically objectionable discoloration may be managed by bleaching, microabrasion, veneering, or crowning. The choice between these treatments depends on the severity of the fluorosis.

Note Acquired Dental Defects

Acquired dental defects include dental caries, attrition, abrasion, and erosion. Dental caries is an infectious process. Attrition is the wearing away of tooth structure due to tooth-to-tooth contact. Abrasion is the wearing away of tooth structure through an abnormal mechanical process. Erosion refers to the loss of tooth structure due to chemical action.

Conclusion

The clinical manifestations of many diseases, either local or systemic, characteristically affect the lips, labial or buccal mucosa, hard palate, soft palate and tonsillar areas, tongue, floor of the mouth, gingivae, and teeth. Knowledge of the more common presentation patterns of a given disease assists the practitioner in determining a diagnosis. It must be remembered, however, that no diagnostic index or outline can fully account for the capriciousness of a disease or the different reactions of an individual host to a disease. Therefore, the evaluation and integration of the clinical appearance and characteristics of a disease, along with the history of development and other appropriate diagnostic findings, are often necessary to determine the final diagnosis.

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6 Radiographic Examination

Diagnostic radiography is an integral part of the clinical process. It is predicated on a careful correlation of patient history and clinical findings. Radiographs should be ordered in those instances in which the clinician anticipates that the expected information obtained will contribute materially to the proper diagnosis, treatment, and

Table 6.1. Elements of an expressed consent in diagnostic dental radiography.

Why are the radiographs necessary?
What alternate diagnostic aids are available?
What risks are inherent in the use of ionizing radiation?
What radiation protection measures will be taken?
What effect the lack of quality dental radiographs may have on the diagnosis, treatment, and prognosis?

prevention of disease. Consequently, the clinician's responsibility in obtaining radiographs involves two major considerations: (1) the clinical decision to order radiographic studies, and (2) the selection of an appropriate number and type of radiographic views necessary to conduct the examination.

In order to maximize diagnostic yield yet minimize the risk of unnecessary exposure, clinicians are responsible for assuring that all radiographs are obtained in accordance with the current standard of care. The type of radiographs, the number of films taken, the date on which they were taken, and the diagnostic data obtained should be documented in the progress notes. Furthermore, since radiographs often represent the only evidence of past dental treatment or disease and serve as the basis for future treatment decisions, they must be retained as part of the patient's permanent record.

Before initiating any radiographic procedures, the clinician is further responsible for obtaining the patient's consent. The consent given by the patient may be implied or expressed. An implied consent is sufficient for commonly performed procedures that have few known risks. When a procedure has perceived or potential risks associated with it, such as the use of ionizing radiation on a child or a pregnant woman, the clinician should receive the guardian's or the pregnant patient's expressed consent (Table 6.1).

Radiographic Examination of the New Patient

Child with Primary Dentition

Open contacts in the primary dentition will often allow the clinician to visually inspect the proximal surfaces of posterior teeth. However, closure of contacts may potentially lead to the development of proximal carious lesions. Therefore, posterior bitewing radiographs are recommended for new patients with primary dentition where the proximal surfaces are not easily visualized or explored.

Child with Transitional Dentition

The incidence of dental caries tends to increase in children with a transitional or mixed dentition as a result of socialization, dietary modifications, and changes in daily oral hygiene procedures. Posterior bitewing radiographs are recommended for new patients with a transitional dentition for the detection of caries. This survey may be augmented with selected periapical views when clinical evidence suggests the presence of an apical pathosis or periodontal disease (rare). Periapical/occlusal views may be appropriate to assess the presence/absence and growth/development of all permanent teeth. A panoramic view with posterior bitewing radiographs is an acceptable alternative.

Adolescent with Permanent Dentition prior to the Eruption of Third Molars

Increased independence and socialization, changing dietary patterns, and decreased attention to daily oral hygiene characterize this age group. Each of these factors may result in an increased risk for dental caries. Although proximal surfaces continue to show caries development, there is a tendency for caries activity to shift from proximal surfaces to surfaces with pits and fissures.

An individualized radiographic examination should consist of posterior bitewing radiographs and selected periapical views. A panoramic view may facilitate the assessment of the presence, position, and stage of development of third molars.

Adult Dentate Patient

Although the incidence of proximal caries in the adult patient population is declining, it is important to assess proximal surfaces in new adult patients for primary and recurrent disease activity (the incidence of root surface caries increases with age, but the usual method of detecting such lesions is by clinical examination). In addition, adult patients may have signs and symptoms of periodontal and/or pulpal disease, or have missing teeth requiring replacement. Therefore, adult dentate patients should have posterior bitewing radiographs and selected periapical films. Routine full mouth radiographs are not indicated unless the patient presents with clinical evidence of generalized dental disease or evidence of extensive past dental care.

Adult Edentulous Patient

Radiographic examination for occult disease in this patient population cannot be justified on the basis of disease prevalence, morbidity, mortality, radiation dose, and cost. However, a full mouth series of periapical radiographs or a panoramic view of the edentulous patient in conjunction with anticipated prosthetic reconstruction is appropriate to assess the presence or absence of impacted teeth, retained roots, bony spicules, residual cysts or infections, developmental abnormalities of the jaws, intrabony tumors, and systemic conditions affecting bone metabolism. In addition, diagnostic imaging provides information on the anatomical location of the mandibular canal, the position of the mental foramen and maxillary sinus, and the relative thickness of soft tissues covering the alveolar ridges.

Radiographic Examination of the Recall Patient

No Clinical Caries and No Evidence of High-Risk Factors for Caries

Child with Primary Dentition

In spite of the general decline in the incidence of dental caries, subgroups of children have a higher caries experience than the general population. The identification of patients in these subgroups may be difficult on an individual basis. Consequently, children with primary dentition with closed posterior contacts that show no clinical caries and that are not at risk for the development of caries benefit from an examination consisting of posterior bitewing radiographs, performed at intervals of 12–24 months. This recommendation is based on evidence that dental caries progress more rapidly in primary teeth (thinner enamel with higher organic components) than in permanent teeth.

Child with Transitional Dentition

The incidence of dental caries generally increases during the transitional period. In addition, the enamel of permanent teeth undergoing posteruptive maturation may facilitate faster progression of carious lesions. Therefore, children with a transitional dentition who show no clinical caries and are not at risk for the development of caries benefit from an examination, consisting of posterior bitewing radiographs, performed at intervals of 12–24 months.

Adolescents

The caries process in the permanent dentition takes about 36 months to progress from initial involvement of the enamel surface to the dentin. Consequently, it is recommended that in adolescents who show no clinical caries and are not at high risk for the development of caries, an examination consisting

of posterior bitewing radiographs be performed at intervals of 18–36 months.

Adult Dentate Patient

Advancing age, changes in the diet, and periodontal and systemic disease in adult dentate patients may increase the risk of dental caries. Consequently, dentate adults who show no evidence of clinical caries and are not at high risk for the development of caries still benefit from an examination consisting of posterior bitewing radiographs performed at intervals of 24–36 months.

Clinical Caries and Evidence of High-Risk Factors for Caries

Children with Primary and Transitional Dentition

Children who are at increased risk for developing dental caries because of such factors as poor oral hygiene, high frequency of exposure to sucrose-containing food, and deficient fluoride intake are more likely to have proximal caries. Because of poor correlation between the histological and radiographic extent of carious lesions, an examination consisting of posterior bitewing radiographs at 6-month intervals is recommended. Such patients remain in the high-risk group until there is no clinical or radiographic evidence of caries.

Adolescents

Adolescents develop proximal carious lesions that may progress rapidly because of factors such as the immature status of the permanent enamel, the behavior of adolescents leading to inconsistent oral hygiene practices, and dietary changes. It is recommended that a radiographic examination consisting of posterior bitewings be performed at 6–12 month intervals. Such patients remain in the high-risk category until there is no clinical or radiographic evidence of caries.

Adult Dentate Patient

The caries process in most adult patients is less likely to progress as rapidly to dentinal involvement as in children or in adolescents. Consequently, it is recommended that a radiographic examination consisting of posterior bitewings be performed at 12–18 month intervals. Such patients remain in the high-risk category until there is no clinical or radiographic evidence of caries.

Radiographic Examination of the Patient with Active Periodontal Disease or a History of Periodontal Treatment

The frequency and type of radiographic examinations for these patients should be based on a clinical examination of the periodontium and documented signs and symptoms of periodontal disease. Patients with 5 mm or greater attachment loss may require vertical bitewing radiographs, where the long axis of the film is oriented in a vertical rather than the normal horizontal direction so the osseous crest is visible on the radiograph.

Radiographic Assessment of Growth and Development

Child with Primary Dentition

Prior to the eruption of the first permanent tooth, radiographic examination to assess growth and development, in the absence of clinical signs and symptoms, is unlikely to yield productive information. Consequently, any abnormality of growth and development suggested by clinical findings should be evaluated radiographically on an individual basis.

Child with Transitional Dentition

Following the eruption of the first permanent tooth, a periapical/occlusal or panoramic examination should be conducted to assess the presence/absence and growth and development of all permanent teeth. This assessment need not be repeated unless dictated by clinical signs and symptoms.

Adolescents

The major concern for patients in this age group is to determine the presence, position, and development of third molars. This can best be accomplished through a radiographic examination consisting of selected periapical films or a panoramic view on a single occasion once the patient is in late adolescence (16–19 years of age).

Introduction to Radiographic Interpretation

In approaching radiographic diagnosis, the first step is to recognize the presence of an abnormality. This is best accomplished by systematically scanning the radiograph. Once an abnormality is encountered, it should be described in general terms and categorized based on location and radiographic appearance. It must, however, be emphasized that in no instance should a clinician arrive at a definitive diagnosis on the basis of radiographic findings alone. The radiographic diagnosis must correlate with the historical profile, physical examination, and, when indicated, with the clinical laboratory data and microscopic analysis.

Radiolucent Versus Radiopaque

As a general rule, lesions in osseous tissue may be divided into three categories: radiolucent, radiopaque, or mixed. Radiolucency indicates some measure of bone destruction. Conversely, radiopacity is more frequently associated with slow-growing lesions that although cause osseous alteration can be regarded as relatively nondestructive. There are exceptions to this concept, especially when the radiopacity is directly associated with a radiolucent area.

A mixed radiolucent/radiopaque appearance is often associated with fibro-osseous lesions, which both resorb and produce bone. In radiopaque and mixed lesions, it is very important to notice the degree of radiopacity. Enamel is the most radiopaque and homogeneous tissue in the human body. Dentin and cementum are as homogeneous as enamel but less radiopaque. Bone, while similar to dentin and cementum in terms of radiopacity, is usually less homogeneous and is characterized by the presence of loose or dense trabeculation.

Unilocular Versus Multilocular

Multiloculation in a single radiolucent area suggests the presence of a slow-growing neoplasm. Round locules presenting a soap bubble appearance are usually noted in well-circumscribed yet locally aggressive tumors like the ameloblastoma or myxoma.

Peripheral Outline

The lesion, whether it is radiolucent or radiopaque, may have a distinct border, or its margins may be rough, irregular, or indistinct. A distinctive opaque lamina or sclerotic border around a radiolucent area suggests a slowly growing noninvasive lesion, as seen in "typical" cystic lesions. A definite, relatively smooth, easily identifiable margin between a radiolucent area and the surrounding bone may be observed when solid granulation tissue develops in bone.

Rough, irregular, or indistinct peripheral margins suggest tissue growth or the spread

of infection beyond the present capacity of the body to wall off or circumscribe. A radiolucent line around a radiopaque area is characteristic of odontomas, particularly the cementomas and the compound composite and complex composite odontomas. A rather easily observed differentiation between an opaque mass and the surrounding bone, even though these tissues blend together, is often associated with a sclerotic bone lesion such as an enostosis.

Trabecular Pattern

Maxillary trabecular pattern is usually quite fine and without any particular directional arrangement. Trabecular bone in the mandible tends to run horizontally and the pattern is larger and more elliptical than those in the maxilla. The normal trabecular pattern, particularly in the mandible, may be altered to show fewer, coarser trabeculae arranged in a horizontal fashion. This gives a so-called "stepladder" appearance to the trabeculae and suggests a disturbance of the hematopoietic system.

The normal pattern may also be replaced by a fine, almost web-like network of bone trabeculae that look like ground glass, as in fibrous dysplasia and various stages of Paget's disease. When the web-like, reticulated, or ground-glass appearance is associated with a reduction in opacity, one must suspect calcium depletion, as may be seen with hyperparathyroidism. Following recalcification, these areas may present a cotton-wool appearance.

Dimensional Changes

Changes in bone size or shape are usually manifest clinically. Expansion of bone apparently takes place as a compensatory mechanism resulting from bone resorption in a directly adjacent area, which may be associated with pressure, chronic infection, or a slow-growing neoplastic lesion. Soft-tissue tumors that have metastasized to bone expand bone inversely to the rapidity of tumor growth. Bone tumors that are true neoplastic osteogenic lesions are inherently expansile. Here the bone growth is due to normal bone formation in an abnormal location at an abnormal rate.

Many expansile lesions cause a characteristic response in the periosteum of the overlying bone; this is called "periosteal reaction." Various appearances have been described such as "onion skin," "sunburst," or "Codman's triangles." Each appearance can be associated with a particular disease entity.

Radiographic Manifestations of Common Conditions

In this chapter, radiographic lesions are introduced as they present in the clinical setting: (1) coronal and pericoronal radiolucent, radiopaque, or mixed lesions (Table 6.2); (2) periapical, intraradicular, or interradicular radiolucent, radiopaque, or mixed lesions (Table 6.3); (3) unilocular and multilocular radiolucent lesions of the jaw bones with distinct borders (Table 6.4); (4) solitary radiopaque or mixed lesions within jaw bones with distinct border (Table 6.5); and (5) multiple or generalized radiolucent lesions with distinct borders, radiolucent lesions with indistinct borders, radiopaque lesions, or mixed lesions within jaw bones (Table 6.6).

Table 6.2. Coronal and pericoronal lesions.

	Condition	Predominant gender	Predominant age	Predominant jaw	Predominant region	Other features
Radiolucent	Caries (Figure 6.1)	M ≈ F	>15 years	Maxilla ≈ mandible	Premolar/molar	
	Periodontal disease (Figures 6.2 and 6.3)	M > F	Adults	Maxilla	Posterior	Many variants of this disease exist
	External resorption (Figures 6.4 and 6.5)	M ≈ F	Adolescents	Maxilla	Anterior	May be iatrogenic (orthodontic)
	Follicular space (Figures 6.6 and 6.7)	M ≈ F	18–25 years	Mandible ≈ maxilla	3rd molar Cuspid	Considered normal if <2.5 mm on periapical radiographs and 3 mm on panoramic radiographs
	Dentigerous cyst (Figures 6.8 and 6.9)	M > F (2 : 1)	20+ years	Mandible	3rd molar	Often encompasses crown from CEJ to CEJ
	Ameloblastic fibroma (Figure 6.10)	M > F (1.5 : 1)	10–15 years	Mandible	Posterior	May extend beyond CEJ
Radiopaque	Compound odontoma (Figure 6.11)	M ≈ F	Second decade	Maxilla	Anterior	Multiple small tooth-like formations
	Complex odontoma (Figures 6.12 and 6.13)	M ≈ F	Second decade	Mandible	Posterior	Homogeneous mass
	Enamel pearl (Figure 6.14)	M ≈ F	Younger patients	Maxilla	Molar	1–3 mm radiopacity in the furcation area
	Pulp stone (Figure 6.15)	M ≈ F	Increases with age	Maxilla ≈ mandible	Premolar/molar	May be solitary or multiple
Mixed	Ameloblastic fibro-odontoma	M ≈ F	Second decade	Maxilla ≈ mandible	Anterior Posterior	Often associated with a missing tooth
	Adenomatoid odontogenic tumor (Figures 6.16a–6.16c)	M < F (1 : 2)	10–20 years	Maxilla	Canine	Often associated with impacted tooth
	Calcifying odontogenic tumor (Gorlin cyst)	M ≈ F	30–50 years	Maxilla ≈ mandible	Premolar/molar	Usually multilocular
	Calcifying epithelial odontogenic tumor (Pindborg tumor)	M ≈ F	10–20 years	Maxilla ≈ mandible	Anterior Posterior	Exists in multiple subtypes



Figure 6.1. Caries.

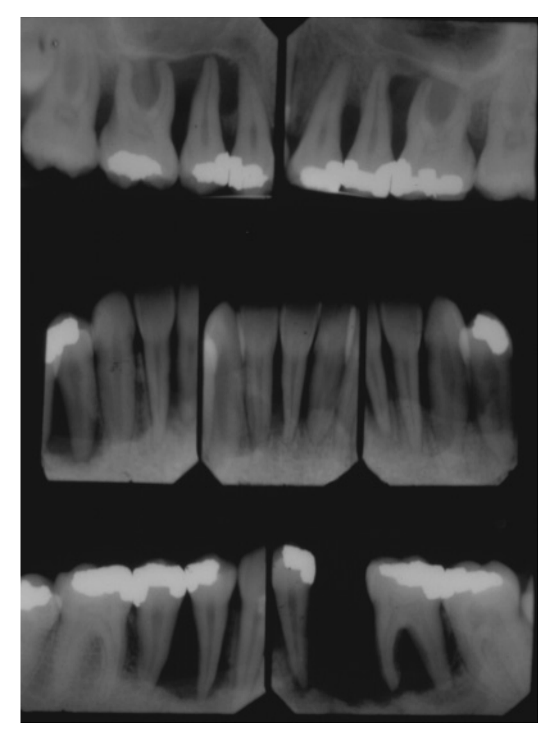


Figure 6.2. Periodontal disease.

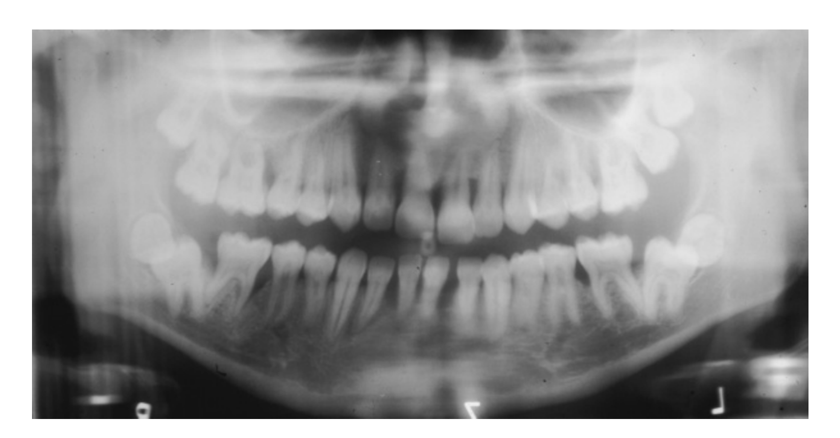


Figure 6.3. Periodontal disease.

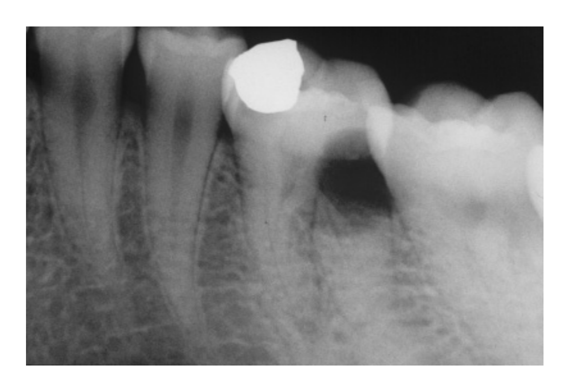


Figure 6.4. External resorption.



Figure 6.5. External resorption.



Figure 6.6. Follicular space.



Figure 6.7. Follicular space.



Figure 6.8. Dentigerous cyst.

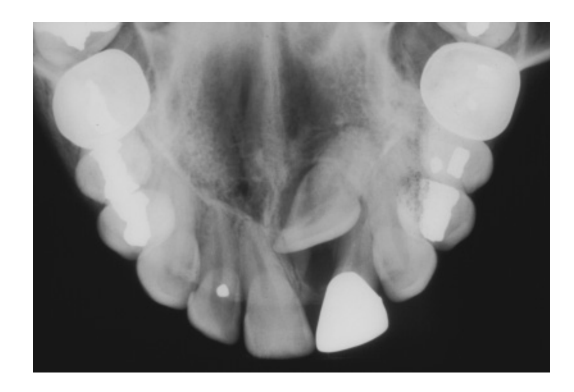


Figure 6.9. Dentigerous cyst.

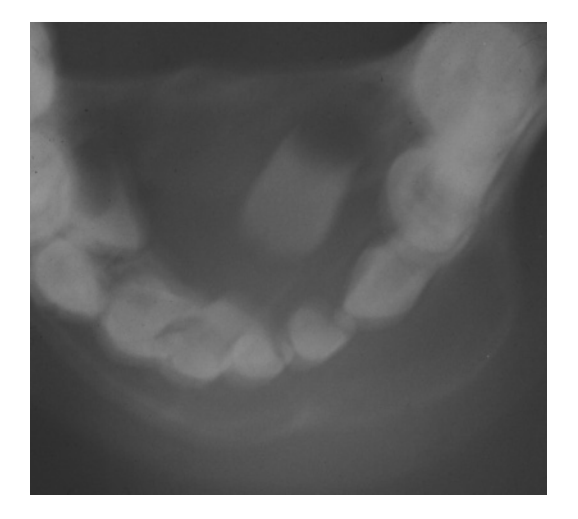


Figure 6.10. Ameloblastic fibroma.



Figure 6.11. Compound odontoma.



Figure 6.12. Complex odontoma.

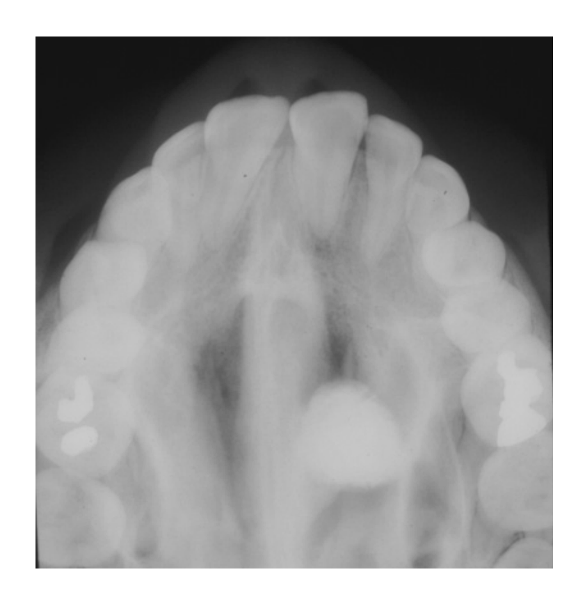


Figure 6.13. Complex odontoma.



Figure 6.15. Pulp stone.

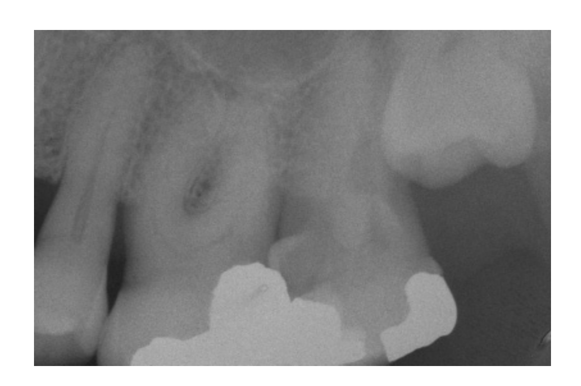
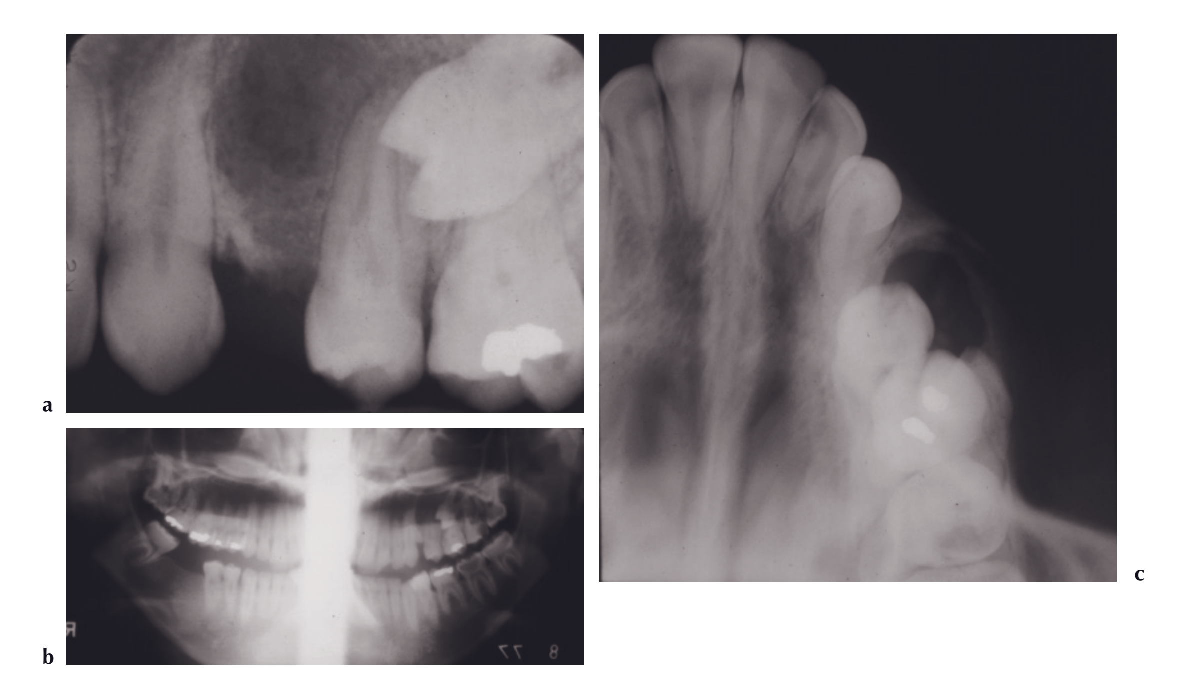


Figure 6.14. Enamel pearl.



Figures 6.16a–c. Adenomatoid odontogenic tumor.

Table 6.3. Periapical, intraradicular, or interradicular lesions.

	Condition	Predominant gender	Predominant age	Predominant jaw	Predominant region	Other features
Radiolucent	Internal resorption (Figures 6.17, 6.18, 6.19, and 6.20)	M > F	Fourth–fifth decades	Maxilla ≈ mandible	Cuspids, 1st and 2nd molars	
	Periapical periodontitis (Figure 6.21), abscess (Figure 6.22), cyst (Figure 6.23), granuloma (Figure 6.24a), scar (Figure 6.24b)	M > F	Third–sixth decades	Maxilla (60%)	Incisors/cuspids	The apex is usually the epicenter of the lesion
	Lateral periodontal cysts	M > F	Fifth–sixth decades	Mandible	Cuspid/premolar	Teeth are usually vital
	Incisive canal cyst (Figure 6.25)	M > F (2.3 : 1)	<20 years of age	Maxilla	Anterior (midline)	Median-palatine cyst is the same lesion located posteriorly
	Traumatic bone cyst (Figure 6.26)	M > F (2 : 1)	Second decade	Mandible	Premolar/molar	Pseudo-cyst, no epithelial lining
Radiopaque	Osteosclerosis	M ≈ F	All ages	Mandible	Canine/premolar	Normal anatomic variant
	Pulp obliteration (Figure 6.27)	M ≈ F	Adults	Maxilla ≈ mandible	Anterior	Consider systemic conditions
	Hypercementosis	M ≈ F	Third–fourth decades	Mandible	Molar	Unknown etiology
Mixed	Condensing osteitis	M ≈ F	All ages	Mandible	Molar/premolar	
	Periapical cemental dysplasia (Figure 6.28)	M < F	Fourth decade	Mandible	Anterior	Three stages; often misdiagnosed as a periapical radiolucency
	Cementoblastoma (Figures 6.29 and 6.30)	M > F	Second–third decades	Mandible	1st molars	Lesion is fused to the roots
	Florid cemento-osseous dysplasia	M < F	Fourth decade	Maxilla ≈ mandible	Found in all regions	Usually multiple lesions



Figure 6.17. Internal resorption.



Figure 6.18. Internal resorption.



Figure 6.19. Internal resorption.

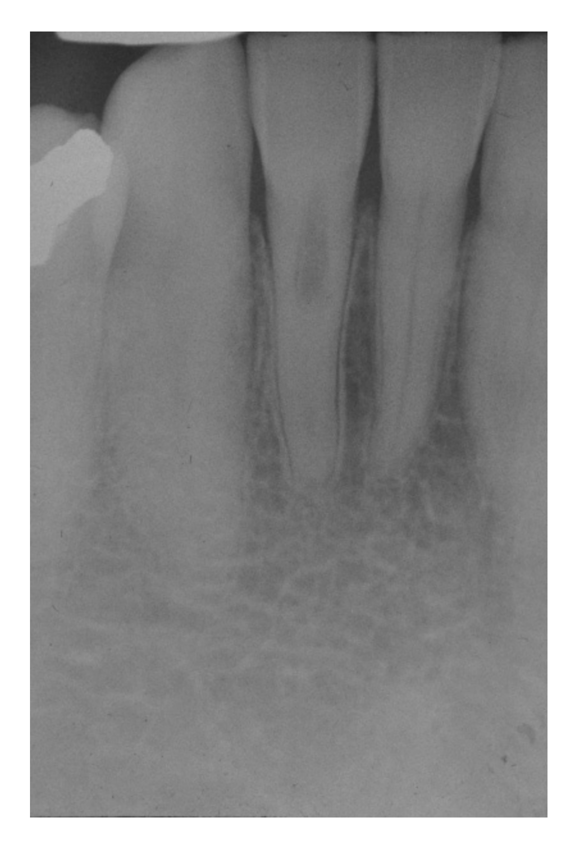


Figure 6.20. Internal resorption.



Figure 6.22. Periapical abscess.



Figure 6.21. Periapical periodontitis.

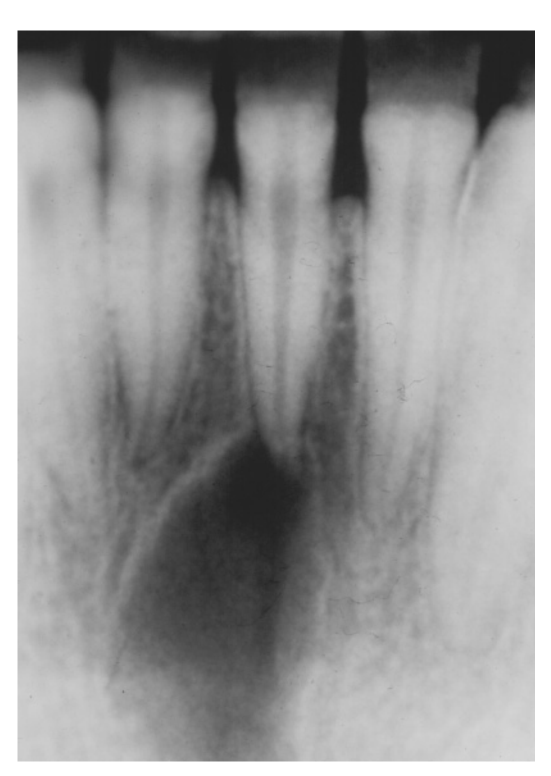


Figure 6.23. Periapical cyst.


a

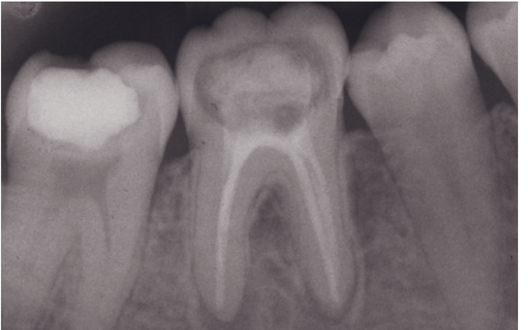

b

Figure 6.24. a. Periapical granuloma. b. Periapical scar.



Figure 6.25. Incisive canal cyst.

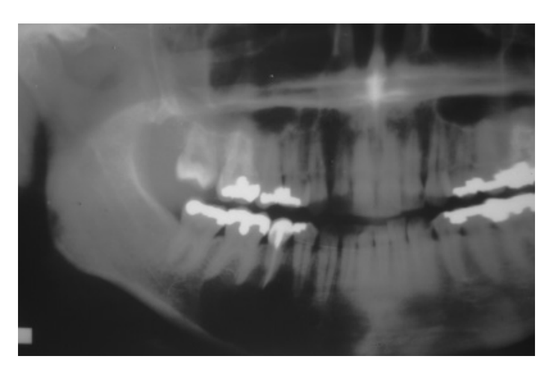


Figure 6.26. Traumatic bone cyst.



Figure 6.27. Pulp obliteration.

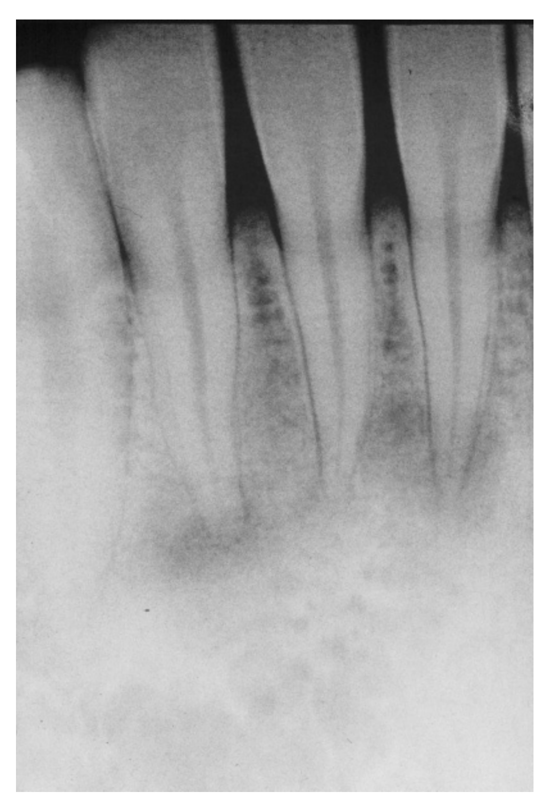


Figure 6.28. Periapical cemental dyslasia.

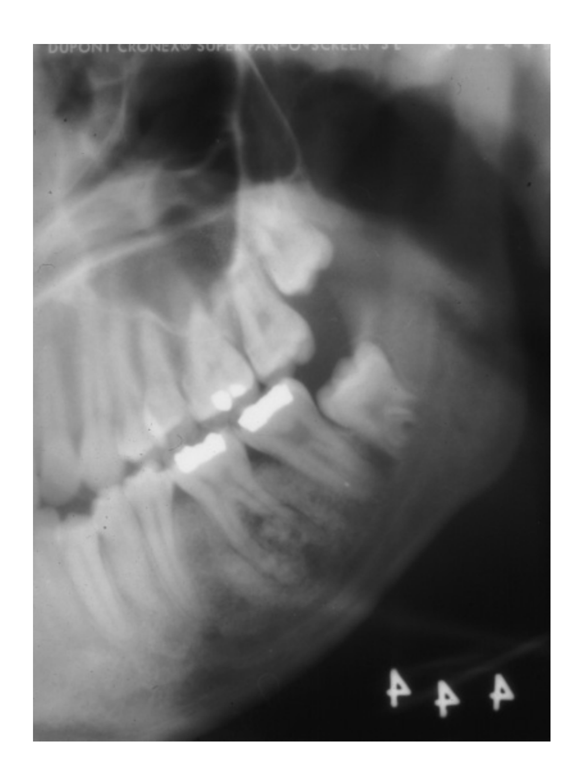


Figure 6.29. Cementoblastoma.

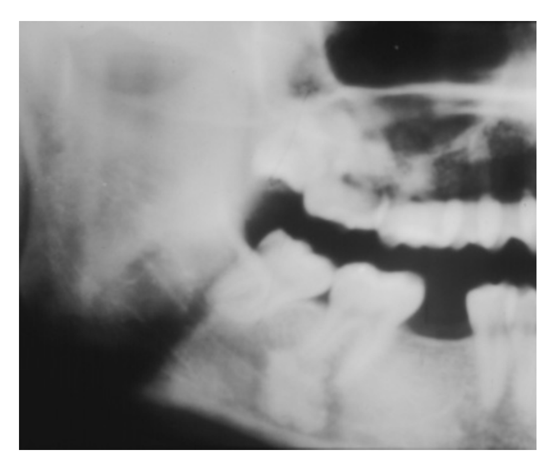


Figure 6.30. Cementoblastoma.

Table 6.4. Unilocular and multilocular radiolucent lesions within the jaw bones.

	Condition	Predominant gender	Predominant age	Predominant jaw	Predominant region	Other features
Unilocular	Salivary gland depression	M ≈ F		Mandible	Posterior	Inferior to the mandibular canal
	Fibrous healing defects (Figures 6.31 and 6.32)	M ≈ F	>30 years	Maxilla	Anterior	
	Hematopoietic bone marrow defect	M < F	Fourth decade and up	Mandible	Posterior	
	Residual cyst (Figure 6.33)	M > F	Older patients	Slightly more in the mandible	All areas	Prior surgical history often noted
	Langerhans cell disease (Histiocytosis-X)	M > F (2 : 1)	Children and adolescents	Mandible	Posterior areas bilaterally	May be isolated or part of a syndrome
Multilocular	Odontogenic keratocyst	M > F (1.5 : 1)	Third–fourth decades	Mandible	Posterior	High recurrence rate
	Ameloblastoma (Figures 6.34 and 6.35)	M > F	Third–fourth decades	Mandible	Posterior	Typical soap bubble appearance
	Central giant cell granuloma (Figures 6.36 and 6.37)	M < F (1 : 2)	<20 years of age	Mandible	Anterior	Generally anterior to 1st molars
	Aneurismal bone cyst (Figures 6.38a–6.38c)	M < F (2 : 3)	<30 years of age	Mandible	Posterior	
	Odontogenic myxoma	M < F	10–30 years of age	Mandible	Posterior	Typical geometric septation
	Central hemangioma (Figures 6.39a–6.39c)	M < F (1 : 2)	Children and toddlers	Mandible	All areas	Auscultation may reveal pulse, bruit



Figure 6.31. Fibrous healing defect.



Figure 6.32. Fibrous healing defect.



Figure 6.33. Residual cyst.

Figure 6.34. Ameloblastoma.



Figure 6.35. Ameloblastoma left maxillary sinus.



Figure 6.36. Central giant cell granuloma.



Figure 6.37. Central giant cell granuloma.

a

b

c

Figures 6.38a–c. Aneurismal bone cyst.



Figures 6.39a–c. Central hemangioma.

Table 6.5. Solitary radiopaque lesions within the jaw bones.

	Condition	Predominant gender	Predominant age	Predominant jaw	Predominant region	Other features
Radiopaque	Exostosis	M ≈ F		Maxilla	Buccal aspect of canines and molars	
	Root fragments	M ≈ F				
	Socket sclerosis	M < F (1 : 2)	Middle-age adults	Maxilla ≈ mandible	Premolar/molar	
	Torus		Middle age	Both	Midpalate	
	Enostosis	M ≈ F	All ages	Mandible	Premolar/molar	Attached to the endosteal surface of the cortex, not detected clinically
	Osteoma	M > F	Older than 40 years of age	Mandible	Posterior/ramus	
	Osteoblastoma	M > F (2 : 1)	Second and third decades	Maxilla ≈ mandible	Posterior/ramus	
	Chondrosarcoma (Figures 4.16a–4.16g)	M ≈ F	Adults	Maxilla ≈ mandible	Maxilla: anterior regions Mandible: condylar head/neck	
	Osteosarcoma (Figures 4.15a–4.15d)	M > F (2 : 1)	Fourth decade and older	Mandible	Mandible: angle and ramus	
Mixed	Cementifying and osseous fibroma	M < F	Adolescents and young adults	Mandible	Molar/premolar	
	Mucous retention phenomenon (maxillary sinus) (Figure 6.40)	M > F	All ages	Sinus		Incidental finding

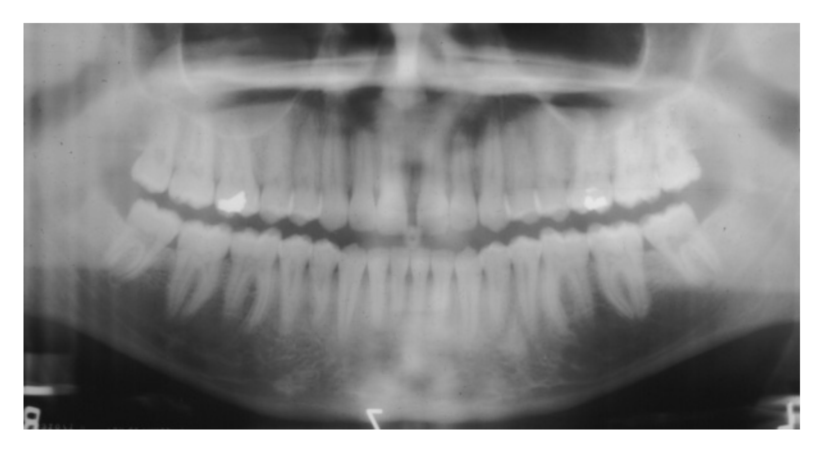


Figure 6.40. Mucous retention phenomenon right maxillary sinus.

Table 6.6. Generalized or multiple lesions within the jaw bones.

	Condition	Predominant gender	Predominant age	Predominant jaw	Predominant region	Other features
Radiolucent (distinct borders)	Multiple myeloma (Figures 6.41a–6.41d)	M > F	35–70 years	Mandible	Posterior/ ramus	Multiple punched out lesions
	Cherubism (Figure 6.42)	M ≈ F	2–6 years	Mandible	Always bilateral	Regresses with age
	Nevoid basal cell carcinoma (Gorlin syndrome)	M ≈ F	5–30 years	Maxilla	Posterior	Jaw manifestation is one of many symptoms
Radiolucent (indistinct borders)	Osteomyelitis	M > F (5 : 1)	>30 years	Mandible (95%)	Posterior	Frequently seen with immunosuppression
	Osteo-radionecrosis (Figure 6.43)	M > F	Elderly	Mandible		History of head and neck irradiation
	Osteopetrosis	M ≈ F	Childhood	Maxilla	Bilateral	Hereditary disorder
	Rickets	M ≈ F	Children	Generalized		
	Hyperparathyroidism (Figures 6.44a–6.44c)	M < F	40–50 years	Generalized		
	Sickle cell anemia	M ≈ F	Children and adolescents	Generalized	Autosomal dominant disease	
Radiopaque	Gardner's syndrome (Figures 4.19a–4.19g)	M ≈ F	Younger patients	Maxilla ≈ mandible	All regions	Hereditary
	Paget's disease (Figure 6.45)	M > F (1.8 : 1)	>40 years	Enlargement of both the maxilla and mandible		
	Fibrous dysplasia (Figures 4.13a–4.13d)	M ≈ F (Albright only female)	Monostatic: second decade Polyostotic: <10 years	Maxilla	Posterior	
Mixed	Metastatic tumors	M ≈ F	>60 years	Mandible	Posterior	
	Florid cemento-osseous dysplasia	M < F	Fourth decade	Maxilla ≈ mandible	Bilateral	



Figures 6.41a–d. Multiple myeloma.

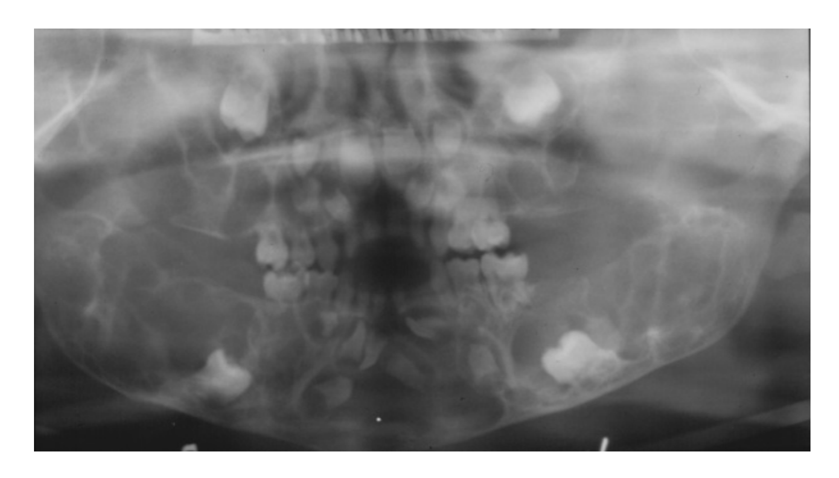


Figure 6.42. Cherubism.

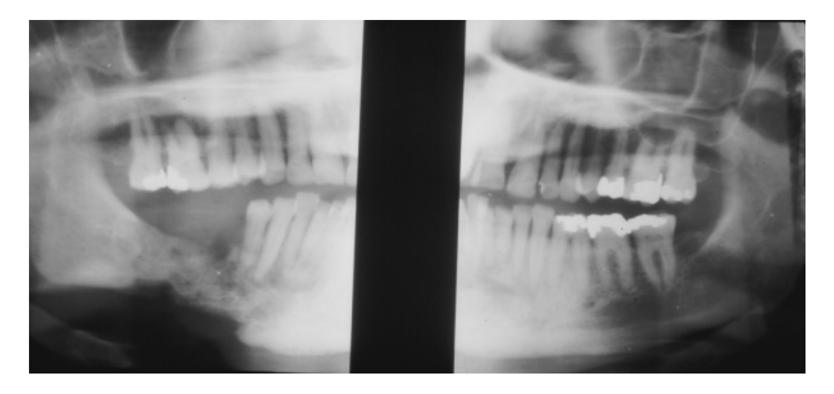


Figure 6.43. Osteo-radionecrosis.

a

b

c

Figures 6.44a–c. Hyperparathyroidism.



Figure 6.45. Paget's disease.

Conclusion

Oral healthcare providers must use clinical judgment to determine the type, frequency, and extent of each radiographic examination. Radiographic imaging should be individualized for each patient and should never be based on administrative or arbitrary requirements such as insurance needs or a fixed time schedule. The goal is to eliminate unnecessary or capricious exposures; that is, follow the dictum of ALARA (As Low As Reasonably Achievable), yet maximize diagnostic yield.

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7 Laboratory Methods

In physical evaluation, clinical laboratory procedures may provide the final clue essential to confirm a diagnosis. They may also lead to the early detection of disorders with vague signs and symptoms, contribute to the discovery of significant, unexpected conditions, or provide a baseline against which response to or the safety of a therapeutic intervention may be measured. Consequently, in some situations, clinical laboratory information may be essential prior to the initiation of therapy. In other instances, it may be an important component of a diagnostic or therapeutic follow-up evaluation. Prior to

ordering laboratory procedures, the clinician should elicit a careful medical history, perform a thorough physical examination, evaluate radiographic studies, and then request the tests from the laboratory that are most likely to either confirm or exclude the provisional diagnosis. A primary organic abnormality may be reflected in specific laboratory findings that may suggest a specific diagnosis or group of diagnoses, prompting the clinician to initiate appropriate therapy, consultation, or referral. This chapter briefly reviews some of the more frequently ordered tests or procedures utilized in medicine and dentistry.

Hematology Screening

The complete blood count (CBC) is an automated test that yields valuable information about red blood cells (RBCs), white blood cells (WBCs), and platelets. It is actually a broad screening panel consisting of several tests utilized to identify numerous disorders such as anemia and infection (Table 7.1). To understand fully the relevance of these tests, it is necessary to examine the clinical significance of elevated or diminished values for the various determinants included in a CBC. However, it should be emphasized that the various blood elements are closely interrelated and individual values must be interpreted in the context of the entire hematological examination.

Table 7.1. Components of CBC.

Red blood cell count	Male: 4.7–6.1 million cells/mcL Female: 4.2–5.4 million cells/mcL
Platelet count	150,000–400,000/mcL
White blood cell count	4,500–10,000 cells/mcL
Hemoglobin	Male: 13.8–17.2 gm/dL Female:12.1–15.1 gm/dL
Hematoctrit	Male: 40.7–50.3% Female: 36.1–44.3%
RBC Indices	
MCV	80–100 fentoliter
MCH	27–31 picograms/cell
MCHC	32–36 gm/dL

Red Blood Cell (RBC) Count

Erythrocytes transport oxygen to all tissues and carbon dioxide to the lungs. Their production is stimulated by hypoxia or anoxia and mediated by erythropoietin. The RBC count expresses the number of RBCs per microliter of whole blood. RBC excess (erythrocytosis) may be caused by dehydration, conditions of low oxygen tension, or polycythemia vera. Reduced numbers of RBCs may occur as a consequence of blood loss (hemorrhage), bone marrow failure, renal disease, hemolysis, leukemia, multiple myeloma, and nutritional deficiencies.

Hematocrit (Hct)

The Hct is the percent volume of packed RBCs in a unit volume of whole blood and provides information about the number and size of red blood cells. An elevated Hct may occur with dehydration, burns, polycythemia vera, and conditions of low oxygen tension (smoking, living at high altitudes, congenital heart disease). A low Hct may be caused by anemia, hemorrhage, bone marrow failure, hemolysis, leukemia, nutritional deficiency, multiple myeloma, and rheumatoid arthritis.

Hemoglobin (Hgb)

Hgb is the oxygen-carrying molecule in erythrocytes and its concentration is reported in grams per deciliter of whole blood. Low Hgb values are usually indicative of either anemia or blood loss.

RBC Indices

The RBC indices consist of three parts. The mean corpuscular volume (MCV), which is a reflection of the relationship between the Hct and the RBC count, is an assessment of the average size of RBCs. RBCs with low, normal, and high MCV values are described as microcytic, normocytic, and macrocytic, respectively. The mean corpuscular hemoglobin (MCH), which is a reflection of the relationship between the Hgb and the RBC count, is an assessment of the average amount of hemoglobin per RBC. RBCs with low, normal, and high MCH values are described as hypochromic, normochromic, and hyperchromic, respectively. The mean corpuscular hemoglobin concentration (MCHC), which is a reflection of the relationship between Hgb and Hct, is an assessment of the hemoglobin concentration in relation to the size of RBCs. The RBC indices provide valuable information used to characterize anemia (Table 7.2).

Table 7.2. Common types of anemia.

Type	Cause
Normocytic normochromic	Acute hemorrhage Hemolysis Aplastic anemia
Microcytic hypochromic	Chronic hemorrhage Iron deficiency Hemoglobinopathies
Microcytic normochromic	Erythropoietin deficiency due to kidney failure
Macrocytic normochromic	Folic acid deficiency Vitamin B_{12} deficiency

White Blood Cell (WBC) Count

There are several types of white blood cells or leukocytes (Table 7.3) that contribute to the WBC count. Neutrophils are the body's predominant phagocytic cell. B-lymphocytes synthesize and secrete antibodies. T-lymphocytes help mediate B-lymphocyte function and have cytotoxic activity against abnormal or virus-infected cells. Monocytes serve as sentinel cells against numerous pathogens and can turn into activated macrophages. Eosinophils and basophils release numerous immunomodulating substances that contribute to the immune response.

The normal WBC count may vary in a particular individual during the course of the day. Conditions of WBC excess (leukocytosis) are associated with infection, inflammatory disease, leukemia, severe emotional stress, or extensive tissue damage. Conditions of WBC deficiency may be observed with bone marrow failure, certain autoimmune disorders, exposure to cytotoxic drugs or chemicals, radiation exposure, or diseases

Table 7.3. WBC differential and associated abnormalities.

WBC type	Normal range (%)	Conditions of excess	Conditions of lack
Neutrophil	40–60	Acute bacterial infection, eclampsia, gout, myelocytic leukemia, rheumatoid arthritis, rheumatic fever, acute stress, thyroiditis, trauma	Aplastic anemia, chemotherapy, influenza, overwhelming bacterial infection, radiation therapy/exposure
Lymphocytes	20–40	Chronic bacterial infection, infectious hepatitis, infectious mononucleosis, lymphocytic leukemia, multiple myeloma, viral infection, recovery from bacterial infection	Chemotherapy, HIV infection, leukemia, radiation therapy/exposure, sepsis
Monocytes	2–8	Chronic inflammatory disease, parasitic infection, tuberculosis, viral infection	NA
Eosinophils	1–4	Allergic reaction, parasitic infection, Hodgkin's disease	NA
Basophils	0.5–1	Acute rheumatic fever, polycythemia vera, myeloproliferative disease	Acute allergic reaction

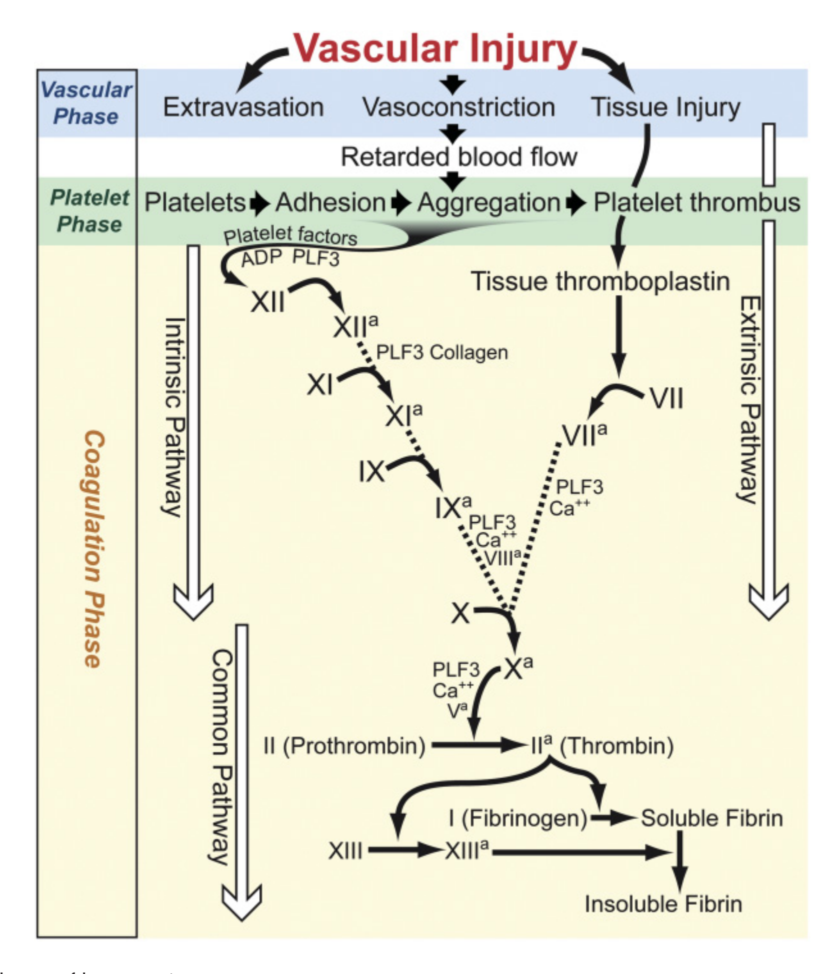


Figure 7.1. Phases of hemostasis.

of the liver and spleen. Minor variations outside the normal range are not significant as long as the differential count on the peripheral blood is normal. The differential count is typically performed if the WBC count is outside the normal range. In a differential count the various types of leukocytes are visually identified and counted on a peripheral blood smear.

Evaluation of Hemostasis

A physiologic mechanism by which the body controls undesirable blood loss is called hemostasis. Adequate hemostasis is dependent on the proper function of its three essential phases: the vascular phase, the platelet phase, and the coagulation phase (Figure 7.1). A discrepancy in one or more of these phases may result in a bleeding disorder, which may be either inherited or acquired.

Vascular Phase

The vascular phase of hemostasis is dependent on the integrity and function of the vasculature. When a blood vessel is damaged, it constricts to reduce blood flow. Examples

of inherited disorders associated with impaired vascular function include hereditary hemorrhagic telangiectasia (HHT) and Ehlers-Danlos syndrome. The most likely acquired disorder of vascular function is allergic purpura. When necessary, a tourniquet test may be performed to clinically assess vessel integrity. In this provocative test, a blood pressure cuff is inflated to maintain pressure halfway between systolic and diastolic for 5 minutes. After deflating the cuff, the skin is allowed to return to its normal color, at which time the number of petechiea visible in a 1-inch-square area on the ventral surface of the forearm is determined (normal: < 20/square inch).

Platelet Phase

The successful formation of a platelet plug is largely dependent on the availability of an adequate number of functioning platelets. The platelet count is a component of the CBC (Table 7.1). A high platelet count (thrombocytosis) may be associated with polycythemia vera, post-splenectomy syndrome, anemia, and certain malignancies. A low platelet count (thrombocytopenia) may be associated with cancer chemotherapy, disseminated intravascular coagulation (DIC), idiopathic thrombocytopenic purpura (ITP), leukemia, and prosthetic heart valves.

Traditional tests to assess the functional capacity of platelets to form a plug include the bleeding time (BT) and the platelet aggregation test. However, today most large hospitals use an automated system, the Platelet Function Analyzer–100 (PFA–100®) to assess platelet function. Inherited disorders of platelet function include von Willebrand's disease and other rare diseases such as Bernard-Soulier syndrome, Glanzmann's thrombasthenia, and storage pool diseases. Acquired disorders of platelet function are far more common than the inherited disorders and may be caused by medications, autoimmune diseases, myeloproliferative diseases, and uremia.

Coagulation Phase

The coagulation phase of hemostasis consists of two distinct pathways, the extrinsic and intrinsic pathways of coagulation (Figure 7.1). The extrinsic pathway is activated in response to tissue damage, while the intrinsic pathway is activated in response to blood vessel wall damage.

Prothrombin Time (PT)

The PT reflects the efficacy of the extrinsic pathway (factors II, VII, X) of coagulation to induce clot formation. Normal PT results range from 11 to 16 seconds. A high PT may be associated with liver disease, bile duct obstruction, vitamin K deficiency, warfarin therapy, malabsorptive disorders, DIC, or deficiencies of factors II, VII, and X.

The PT is utilized not only to assess an undiagnosed bleeding disorder but also to monitor the therapeutic effect of oral anticoagulants (e.g., warfarin). However, testing reagents used to determine PT vary from laboratory to laboratory, which precludes direct comparisons between results. Efforts to solve the problem of variability among testing reagents culminated with the adoption, by the World Health Organization (WHO), of the International Normalized Ratio (INR) system. The INR is the PT ratio that one would have obtained if the WHO reference reagent had been used to perform the PT on the sample.

For most clinical scenarios in which an anticoagulant effect is indicated, a moderate-intensity anticoagulant effect with a targeted INR of 2.0–3.0 is appropriate. Anticoagulant therapy for patients with prosthetic heart valves is optimal when the INR is between 3.0 and 4.0. With a less intense therapeutic range (INR 2.0–3.0), the risk of bleeding is reduced significantly compared with the greater intensity protocols. Abnormal bleeding that occurs when the INR is below 3.0 is typically associated with an obvious underlying cause such as concomitant use of antithrombotic agents, or

serious coexisting systemic conditions such as renal insufficiency, anemia, the presence of a structural defect (such as a polyp in the colon), or bladder tumor.

Prior to an oral surgical procedure, an assessment of the patient's level of anticoagulation is essential to ensure the INR value is within the targeted therapeutic range. Routine dental care (including simple oral surgical procedures) can be delivered with an INR of 4 or less, provided local hemostatic measures are used. For cases in which the INR exceeds 4, it is the responsibility of the patient's physician to make dosage adjustments. Warfarin has a plasma half-life of 36–42 hours; consequently, any change in the dosage will require about 2 days to be reflected in the INR value. Once an acceptable therapeutic range has been achieved, one may perform the procedure. Local anesthesia should be administered cautiously to minimize the risk of hematoma formation. Further measures to augment hemostasis include minimizing surgical trauma, obtaining primary closure when possible, placing sutures to stabilize the tissues, and using local hemostatic agents.

Activated Partial Thromboplastin Time (aPTT)

The aPTT reflects the efficacy of the intrinsic pathway of coagulation to induce clot formation. Normal aPTT results range from 25 to 35 seconds. An elevated aPTT may be associated with numerous disease states, for example, cirrhosis, DIC, hypofibrinogenemia, malabsorption, von Willebrand's disease, lupus, or deficiency of coagulation factors VIII, IX, or XII. Such deficiencies are typical of hereditary coagulation disorders. The aPTT also reflects the level of anticoagulation produced by heparin therapy.

Traditional unfractionated heparin therapy is provided intravenously and thus requires hospitalization. Frequent monitoring is required to maintain the ratio of the patient's aPTT to the mean control aPTT within a defined range. For anticoagulation, an aPTT of 1.5–2.5 times normal is usually desired. When deemed necessary by the physician, heparin therapy may be discontinued approximately 4 hours before oral surgical procedures. Surgery is performed utilizing local anesthesia, atraumatic surgical technique, application of local hemostatic agents, and careful suturing. Heparin therapy is usually reinstated on the day of surgery if there is no active postsurgical bleeding.

The aPTT is also used to monitor the therapeutic efficacy of low molecular weight heparins (LMWHs). LMWHs have a predictable bioavailability and can be self-administered. Their use may serve as a more cost-effective approach to manage patients on warfarin for whom anticoagulation level adjustment is required prior to undergoing extensive surgery. Prior to the availability of LMWHs, the patient was admitted to the hospital, where traditional heparin therapy was substituted for warfarin therapy (the so-called heparin window). Typically on the fifth day of hospitalization, the patient would undergo the surgical procedure, at which time the warfarin would be reintroduced. Once the therapeutic level of warfarin is reestablished, the patient is released. By prescribing an LMWH in lieu of traditional heparin, the physician can now prepare the patient for the anticipated surgery on an outpatient basis.

Biochemical Tests

From the analysis of large numbers of biochemical profiles, certain patterns emerge that are sufficiently characteristic to suggest a specific diagnosis or group of diagnoses. This is analogous to the pathologist's recognition of tissue patterns when examining specimens and may be thought of as a "biochemical biopsy." A primary organic abnormality is typically reflected in the findings of certain tests, and abnormalities detected by other tests help to arrive at a more specific

Table 7.4. Biochemical tests.

Test	Normal values
Liver panel	
ALT	N/A
AST	10–34 IU/L
ALT	44–147 IU/L
Bilirubin	
Total	0.3–1.9 mg/dL
Direct	0.0–0.3 mg/dL
TP	6.0–8.3 g/dL
Albumin	3.4–5.4 g/dL
A/G	1.5–2.5/1
LDH	105–333 IU/L
Fasting blood glucose	60–100 mg/dL
HbA1c	< 5%
Lipid panel	
Cholesterol	< 200 mg/dL
LDL	< 100 mg/dL
HDL	> 60 mg/dL
Triglycerides	< 150 mg/dL
BUN	7–20 mg/dL
Uric acid	3.0–7.0 mg/dL
Creatine	0.8–1.4 mg/dL
Calcium	8.5–10.2 mg/dL
PO_4	2.4–4.1 mg/dL

IU = International unit
L = liter
dL = deciliter
mg = milligram
g = gram

diagnosis and may signal secondary involvement of other systems. Some of the more common laboratory tests to diagnose specific abnormalities are discussed (Table 7.4). Many of these tests are ordered in groups or panels. For example a liver panel consists of ALT, ALP, AST, bilirubin, albumin, and total protein.

Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST)

ALT is an enzyme found predominately in the liver and to a lesser degree in the kidneys, heart, and muscles. AST is an enzyme found predominantly in the liver and heart, and to a lesser extent in other organs. High ALT levels are specific for liver disease or exposure to hepatotoxic drugs. High AST levels may be associated with liver disease and other conditions such as acute pancreatitis, acute renal failure, myocardial infarction, severe burns, muscle trauma, surgery, or progressive muscular dystrophy.

Alkaline Phosphatase (ALP)

ALP is a ubiquitous enzyme found predominately in the liver, bone, and placenta. Various isoenzymes of ALP exist, each correlating with a specific tissue type. High ALP levels may be associated with liver disease, anemia, biliary obstruction, Paget's disease, bone healing, leukemia, or pregnancy. Low ALP levels may be associated with a nutritional deficiency.

Bilirubin

Bilirubin is a by-product of hemoglobin metabolism derived mainly from physiological red cell destruction in the reticuloendothelial (RE) system. The water-insoluble plasma bilirubin is transported to the liver, where it is conjugated with glucoronide and eliminated into the bile. Conjugated bilirubin is referred to as direct bilirubin and unconjugated bilirubin is referred to as indirect bilirubin. The typical bilirubin assay determines both the total bilirubin and direct bilirubin values, allowing for a deductive determination of the indirect bilirubin level. High indirect bilirubin levels are associated with hemolytic anemia, sickle cell anemia, pernicious anemia, transfusion reaction, and Gilbert's disease. High direct bilirubin levels are associated with liver disease and bile duct obstruction.

Total Protein (TP), Albumin, and Albumin/Globulin (A/G) Ratio

Dietary proteins are hydrolyzed in the alimentary canal into amino acids and transported to the liver and the RE system for the

synthesis of body proteins. Blood proteins are classified as either albumins or globulins. Albumins are produced by the liver and are the predominant proteins in the plasma. They serve to transport many small molecules (e.g., hormones, vitamins, drugs, ions) in the blood and are also essential for the maintenance of the osmotic pressure of the blood. Globulins are further classified as being alpha-1, alpha-2, beta, and gamma globulins. The gamma globulins include the various antibody types (M, G, A, E) and are synthesized by the RE system.

TP is a composite of the albumin plus globulin values. An elevated albumin value may occur with dehydration, while an elevated TP may be associated with chronic infection or inflammation, multiple myeloma, and Waldenström's disease. Low albumin and/or TP values may be associated with liver disease, renal disease, malabsorptive disorders, malnutrition, and severe burns. A reversed albumin/globulin ratio may be associated with malnutrition, chronic liver disease, and hypergammaglobulinemia.

Lactate Dehydrogenase (LDH)

LDH is an intracellular enzyme present in virtually all metabolically active cells, with its highest concentration found in erythrocytes, heart, liver, kidneys, and skeletal muscle. High levels of LDH are usually associated with destructive processes, that is, myocardial infarction, cerebrovascular accident, hemolytic anemia, hepatitis, pancreatitis, or muscle injury.

Blood Glucose and HbA1c

Most carbohydrates are metabolized into glucose and stored in the liver as glycogen. The utilization of glucose by the body is under the influence of insulin, which facilitates its transfer across cell membranes. The most accurate determination of the blood glucose level is obtained by having the patient fast for at least 6 hours prior to undergoing the test. Elevated blood glucose (hyperglycemia) is usually associated with either impaired glucose tolerance or diabetes mellitus. Low blood glucose (hypoglycemia) is usually associated with an inadequate dietary intake of sugar, exogenous insulin overdose, or therapy with oral hypoglycemic agents.

The HbA1c is a test used to monitor blood glucose control over an extended period. It represents the percentage of hemoglobin in red blood cells that become glycosylated (that is, chemically linked to glucose) during their life span. HbA1c reflects glucose levels in the blood over the previous 6–12 weeks prior to the test. An elevated HbA1c is usually indicative of poorly controlled diabetes mellitus, which is associated with an increased risk of developing eye disease, kidney disease, heart disease, stroke, and nerve damage.

Lipid Panel (Cholesterol, HDL, LDL, VLDL, Triglycerides)

Lipid panel testing is a valuable tool to assess cardiovascular risk. Cholesterol is an essential body constituent required for cell membrane formation, the synthesis of bile acids, and the synthesis of steroid hormones. Water insoluble, it is carried by various lipoproteins (very low density lipoproteins [VLDLs], low density lipoproteins [LDLs], and high density lipoproteins [HDLs]). Triglycerides are compounds used to move fatty acids (formed when fats or oils are consumed) through the blood. Fatty acids may be metabolized for energy or stored (as fat) for later use.

The cholesterol value is a summation of the VLDL, LDL, and HDL and should ideally be less than 200 mg/dL. Elevated cholesterol levels may be associated with a high-cholesterol diet, hypercholesterolemia, hypothyroidism, diabetes mellitus, and nephrotic syndrome. Low cholesterol levels may be indicative of nutritional deficiency,

hyperthyroidism, liver disease, and pernicious anemia. Of all the components of the lipid panel, the LDL appears to be the strongest predictor of cardiovascular risk.

Blood Urea Nitrogen (BUN)

BUN is the chief nitrogenous by-product of protein metabolism. It is produced in the liver and excreted primarily by the kidney. The BUN is commonly ordered to assess renal function. An elevated BUN may be associated with renal disease, cardiovascular disease, urinary tract obstruction, dehydration, and excess protein ingestion.

Uric Acid

Uric acid is a metabolite of nucleic acid degradation, which occurs mainly in the bone marrow and organs of high metabolic activity such as the liver. It is the end product of purine metabolism and is excreted primarily by the kidneys. High uric acid levels may be associated with gout, diabetes mellitus, alcoholism, hypoparathyroidism, lymphoproliferative or myeloproliferative disorders, and renal disease.

Creatinine

Creatine, a natural amino acid derivative, is synthesized in the liver, kidney, and pancreas and is supplied exogenously through the diet (meat, fish). Cells with high-energy requirements such as skeletal muscle use creatine in the form of phosphocreatine, which serves as a phosphate donor to generate ATP from ADP. Serum concentrations of creatinine, a waste product of creatine, reflect creatine utilization, which is proportional to the body's muscle mass, and its excretion by the kidney. Elevated levels may be associated with renal disease, muscular dystrophy, rhabdomyolysis, and acromegaly.

Calcium

Over 90% of calcium in the body is found in the skeleton and teeth. Although the calcium concentration of the extracellular fluid is relatively small, its level is precisely regulated by parathyroid hormone, total protein, vitamin D, and calcitonin. Elevated calcium levels (hypercalcemia) may be associated with hyperparathyroidism, Paget's disease, malignancies, hyperthyroidism, drug therapy, excess calcium ingestion, or sarcoidosis. Low serum calcium levels (hypocalcemia) may be a sign of vitamin D deficiency, pregnancy, renal disease, hypoparathyroidism, or drug therapy.

Phosphorus

About 85% of the total phosphorus is combined with calcium in the skeleton and the rest is distributed as phosphate (PO_4) ion in the blood and other tissues. PO_4 is involved in most metabolic processes. The parathyroid hormone mediates an increased rate of absorption of both calcium and phosphorus and regulates phosphate loss and calcium retention by its effect on renal tubular reabsorption. Elevated PO_4 levels (hyperphosphatemia) may be associated with renal disease, hypoparathyroidism, bone metastasis, liver disease, sarcoidosis, or hypocalcemia. Low PO_4 levels (hypophosphatemia) may be associated with hyperparathyroidism, hypercalcemia, and inadequate dietary intake of PO_4 or vitamin D.

Tissue Studies

There are striking similarities among the many lesions affecting oral tissues. It is essential in the differential diagnostic process that all possibilities are considered before

making a definitive diagnosis. In some instances the history of a given lesion, combined with the characteristic clinical, radiographic, and laboratory findings, may be sufficient to confirm the clinical impression. However, at other times a biopsy may be required to arrive at a specific diagnosis. This is especially true for oral soft-tissue lesions. As a rule, a biopsy is indicated in the management of any suspicious lesion that either persists or does not respond to conventional therapy within 7–14 days.

Scalpel Biopsy

Histologic assessment of a biopsy specimen represents the gold standard for diagnosing any suspicious oral lesion. An excisional biopsy is the technique of choice when a lesion is relatively small. The lesion is excised in its entirety. An incisional biopsy is indicated when a lesion is too large for easy excision. A pie-shaped wedge is removed to include both normal and abnormal tissue. In some instances several specimens may have to be obtained to allow for an adequate microscopic evaluation. In performing a biopsy, certain guidelines should be followed (Table 7.5). Finally, if the practitioner is uncomfortable performing a biopsy or believes the lesion in question is malignant, he or she must promptly refer the patient for appropriate management.

Table 7.5. Guidelines for performing a biopsy.

Do not inject anesthesia directly into the lesion.
Do not crush or macerate the tissue.
Orient the specimen properly.
Immediately place the specimen in an appropriate preservative.
Provide the pathologist with an adequate history, clinical description to include exact location, photographs, and radiographs.
If the initial biopsy fails to confirm, or is inconsistent with, the clinical impression, a repeat biopsy and close monitoring of the lesion must be performed.

Brush Biopsy

Some authorities have expressed concern that small mucosal lesions that appear to be harmless may be overlooked and thus not undergo appropriate follow-up scrutiny (i.e., scalpel biopsy). This reality has led some to advocate the use of the brush biopsy technique to assess the malignant potential for such innocent-appearing lesions. A refinement of the technique is marketed as the OralCDx™ kit (OralScan Laboratories, Suffern, NY). The kit consists of a sealed and sterile oral brush biopsy instrument, a precoded glass slide and matching coded test requisition form, two single-use alcohol/cardovax fixative packs, an alcohol/polyethylene glycol fixative pouch, and a preaddressed container in which to submit the contents.

Proper utilization of the sampling instrument incurs minimal discomfort or bleeding and assures the attainment of an adequate biopsy sample of all three epithelial layers (superficial, intermediate, and basal) of the lesion. The test requisition form includes space for demographic data such as the patient's age, sex, and history of tobacco use, as well as for the location and clinical description of the lesion. All OralCDx specimens are stained in accordance with a modified Papanicolaou method prior to being scanned by a computer. All positive specimens are assessed by an oral pathologist.

The OralCDx is specifically promoted and marketed as a tool for assessing lesions the dentist would likely not routinely choose to biopsy. It is not generally recommended to assess suspicious lesions. Positive findings from OralCDx mandate a definitive scalpel biopsy, and negative results may result from inadequate sampling. The utility of the technique should be limited to that of a bridging procedure for the patient who is either averse to undergoing a biopsy or likely to not return

Table 7.6. Guidelines for performing exfoliative cytology.

Scrape a moist tongue depressor gently over the entire surface of the lesion.
Spread the collected cells evenly on a glass slide.
Spray the slide with an appropriate fixative.
Submit the specimen to the pathologist, along with an adequate history, a clinical description to include exact location, and any photographs.

Table 7.7. DIF desquamative autoimmune conditions.

Condition	DIF characteristics
Pemphigus	Intercellular reticulate deposits of IgG and C3
MMP	Linear deposition of IgG and C3 in area of dermo-epidermal basement membrane zone
Linear IgA dermatosis	Linear deposits of IgA at the basement membrane

for the 2-week follow-up assessment of the initial lesion.

Exfoliative Cytology

Exfoliative cytology may be defined as the microscopic examination of cells harvested from the surface of a lesion (as opposed to the examination of tissue blocks in biopsy). The procedure is inexpensive, quick, easy, and painless (Table 7.6). Properly used, oral exfoliative cytology may be useful in diagnosing viral infections, candidiasis, and, in rare instances, changes suggesting dysplasia or squamous cell carcinoma. Any findings suggestive of malignancy must be either biopsied or promptly referred for management.

Direct Immunofluorescence

Bullous or blistering autoimmune diseases are divided into two general groups, pemphigus and pemphigoid. Pemphigus is characterized by intraepithelial splitting leading to the development of fragile thin-walled blisters. The pemphigoid group is characterized by subepithelial splitting and the development of thicker-walled blisters. This group encompasses bullous pemphigoid, mucous membrane pemphigoid, and linear IgA dermatosis. For all of these conditions, routine histologic analysis of biopsy specimens is often insufficient to establish the diagnosis.

Direct immunofluorescent (DIF) testing is useful in diagnosing a variety of the bullous or desquamative autoimmune conditions. To be diagnostic, the specimen must entail intact epithelium and should be large enough to be easily split into two separate specimens. One specimen is submitted in formalin for routine histologic interpretation. The specimen submitted for direct immunofluorescence must be kept moist on saline-soaked gauze or filter paper and immediately delivered to the laboratory. If immediate delivery is not feasible, appropriate transport media (e.g., Michel's solution) is available from the biopsy service. Fluorescent tagged anti-human immunoglobins are applied to the specimen to determine the presence of specific tissue-bound autoantibodies such as immunoglobulin G (IgG), immunoglobulin M (IgM), immunoglobulin A (IgA), complement factor 3 (C3), and fibrin. Specific autoantibodies localization correlates well with specific bullous or desquamative autoimmune conditions (Table 7.7).

Indirect Immunofluorescence

Indirect immunofluorescence (IIF) is used to detect circulating serum autoantibodies. For pemphigus disease, monkey esophagus is used as the substrate to test for the presence of IgG autoantibodies. For bullous disorders, a sodium chloride split skin technique is used to define the specific diagnosis.

Gram's Staining Method

The gram stain is a valuable and time-tested method used to assess an infected body fluid

Table 7.8. Gram's staining method.

Crystal violet wash for 1 minute.
Rinse with water.
Gram's iodine wash for 1 minute.
Rinse with water.
Decolorize with acetone and alcohol.
Rinse with water.
Counterstain for 10–30 seconds with 2.5% safranin.
Wash and dry.

or tissue (Table 7.8). It provides preliminary information that helps in choosing the best antibacterial agent to prescribe while awaiting definitive identification by a culture. The gram stain technique is a simple laboratory procedure that produces diagnostic slides requiring only an oil emersion microscope for interpretation. It separates microorganisms into two general categories: gram-negative organisms, which appear red following discoloration by alcohol and counterstaining with safranin; and gram-positive microorganisms, which preclude the extraction of the crystal violet-iodine complex by alcohol and appear deep purple in color. Based on their morphologic appearance, microorganisms responsible for bacterial infections may also be described as cocci or bacilli.

Microorganisms may also be aerobic, anaerobic, or facultative. Aerobic organisms are those requiring oxygen to survive, and anaerobic organisms are those that must avoid oxygen to survive. Some organisms may be facultative and survive either with or without oxygen. Clinical clues to the presence of anaerobes include the formation of abscesses, the presence of tissue necrosis, the production of gas within the tissues, the presence of a foul odor (the absence of an odor does not rule out anaerobes), and a failure to grow bacteria on an aerobic culture media.

Conclusion

A primary organic abnormality is typically reflected in the findings of laboratory and tissue studies that are sufficiently characteristic to suggest a specific diagnosis or groups of diagnoses and prompt the clinician to initiate appropriate therapy, consultation, or referral.

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8 Putting It All Together: Introduction to Treatment Planning

Table 8.1. Examples of problems/diagnoses requiring consideration.

Systemic problems	Endodontic problems
Restorative problems	Irreversible pulpitis
Reversible pulpitis	Necrotic pulp
Primary or recurrent caries	Acute apical periodontitis
Lost restoration	Acute apical abscess
High restoration	Periodontal problems
Improper proximal contact	Gingival abscess
Tight contact	Periodontal abscess
Open contact	Necrotizing ulcerative gingivitis
Overhang	Postoperative problems
Cracked tooth syndrome	Pain
Trauma	Root surface sensitivity
Injuries affecting hard tissues	Bleeding
Infraction	Injection
Uncomplicated crown fracture	Swelling
Complicated crown fracture	Lost dressing and/or sutures
Crown-root fracture	Increased tooth mobility
Root fracture	Sequestra
Injuries affecting attachment	Oral surgical problems
apparatus	Nonrestorable tooth
Concussion	Pericoronitis
Subluxation	Postoperative problems
Extrusion	Pain
Lateral displacement	Bleeding
Intrusion	Alveolar osteitis
Avulsion	Nerve injury
Prosthodontic problems	Infection
Missing teeth	Air emphysema
Complete dentures	Soft tissue injury
Fractured artificial teeth	Jaw fracture
Fractured denture base	Oral medicine problems
Deficient posterior palatal seal	Traumatic ulcers
Removable partial dentures	Recurrent aphthous stomatitis
Fractured artificial teeth	Herpes simplex virus infection
Fractured base or flange	Oral candidiasis
Fractured metallic connector	Lichen planus
Loss of abutment or other teeth	Erythema multiforme
Implant failure	Xerostomia
Fixture fracture	Burning mouth syndrome
Fastener failure	Suspected malignancy
	Temporomandibular disorders
	Socio-economic problems

As discussed in chapter 1, the clinical process is sequentially divided into three main components: (1) data collection, (2) establishment of the problem (problem list or diagnoses), and (3) development, presentation, and implementation of the treatment plan (Figure 8.1). The intent of this chapter is to provide the fabric for the treatment planning process.

Data collection is the indispensable first step in initiating the clinical process. Interpreting and correlating the database in the light of principles gained from basic biomedical and clinical sciences will lead to the establishment of coherent, defendable, relevant, and timely diagnoses (Table 8.1), which provide the basis for the development of preventive and therapeutic strategies.

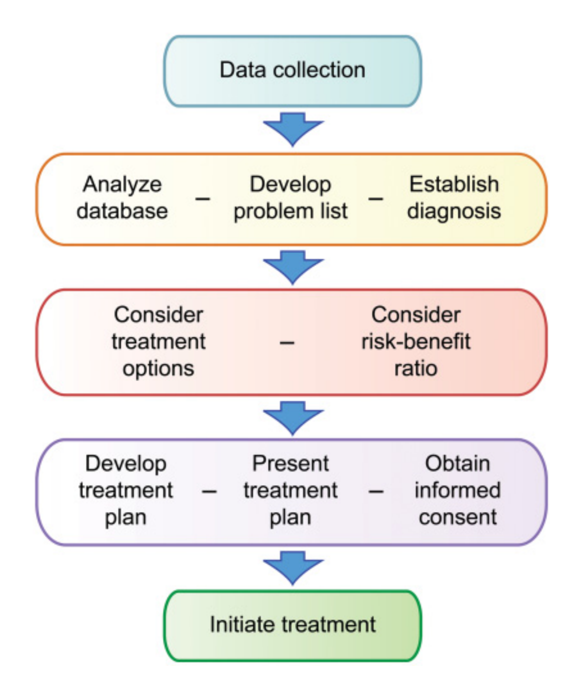


Figure 8.1. Clinical process.

Rational Approach to Treatment Planning

Within the concept of TQM, as a treatment plan deviates from optimal design and implementation, its quality (value, outcome) decreases at an accelerated rate. Consequently, in considering preventive and therapeutic options the clinician must consider not only disease-related variables, but such other factors as the availability of material resources (e.g., facilities and equipment); human resources (e.g., the clinician's own knowledge and technical skills, the availability of an adequate number of qualified support personnel, and a cooperative patient [a patient physically and/or psychologically able to undergo and respond to dental care]); and organizational resources (access to consultants).

The above variables clearly affect outcome and mandate different solutions for identical problems and, at times, may even preclude satisfactory resolution of a specific problem in a given setting. Furthermore, both the clinician and the patient must take into consideration that the treatment of most diseases is predicated on the premise that healing is promoted by modifying the environment of tissues. This, however, requires time. Even if all preventive and treatment procedures were to be implemented on the same day, it would not provide for an immediate optimal healing environment. An effective approach to deal with this problem is to manage disease/illness in phases (Figure 8.2).

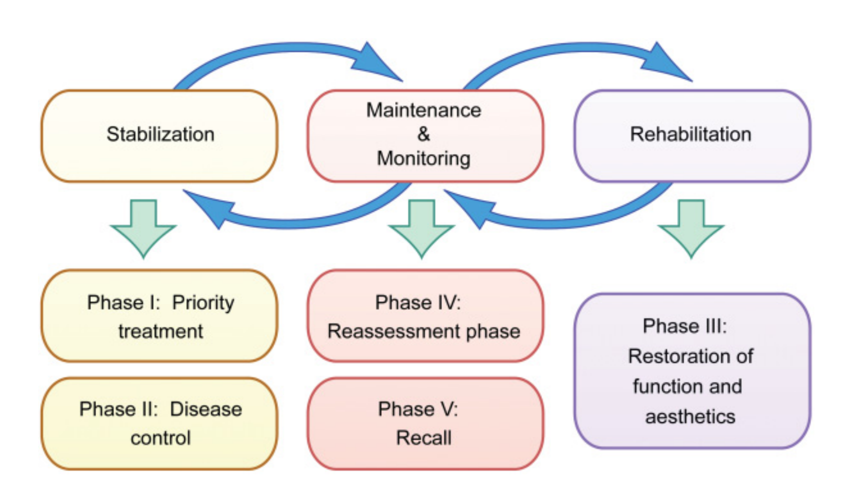


Figure 8.2. Phases of managing disease/illness.

Phase I: Priority Treatment

The goal in Phase I is to deal with problems such as pain, infection, trauma, or other issues of immediate concern requiring priority management. The treatment of acute periodontal and endodontic problems, extraction of a symptomatic tooth with hopeless prognosis, biopsy of a suspicious lesion, excavation of caries approaching the pulp and the placement of a temporary restoration, the management of acute mucosal lesions, and repairing a fractured prosthesis are all procedures that may be performed in this phase.

Phase II: Disease Control

The goal in Phase II is to arrest or manage disease processes. Procedures for controlling rampant caries, chronic periodontal problems, chronic pulpal problems, elective surgical procedures, preliminary elimination of occlusal disharmonies, and management of the patient's chief complaint (if not addressed in Phase I) are the activities that are appropriate for this phase.

Phase III: Restoration of Function and Esthetics

The goal in Phase III is to restore function and improve esthetics. Procedures may include restoration of the remaining carious teeth, replacement of defective restorations, and replacing missing teeth.

Phase IV: Reassessment

The goal in Phase IV is to confirm that all problems have been addressed and that no new problems emerged, and to establish an appropriate recall interval for continued monitoring and maintenance of patients' oral health. A patient satisfaction questionnaire concerning patients' experiences and impressions of treatment and of the treatment environment can provide a mechanism for continued improvement in patient care.

Phase V: Recall

The goal in Phase V is to monitor patients for new or recurrent problems and to implement appropriate corrective and preventive care. Within the concept of TQM, the recall visit also provides an opportunity to evaluate outcomes: (1) the success or failure of preventive and therapeutic strategies; (2) the success or failure of behavior modification characterized by enhanced oral health–related knowledge; and ultimately, (3) improved oral health of the patient.

Presentation of the Treatment Plan

Once a treatment plan (with alternative treatment options) has been developed, it must be communicated to the patient or guardian in a clear and concise manner. The purpose of the case presentation is to provide clinicians with the opportunity to discharge their "duty of disclosure" and for patients to obtain the necessary information essential to exercise their right of "self-determination."

Informed Consent

In order to make informed choices, patients need to know their rights as patients. Those rights include the doctrine of informed consent. Before undergoing any oral health-care–related procedure, patients are legally entitled to an explanation (in terms and phrases that they understand) of the plan so that they can give what is called "informed consent." In those cases in which the patient is unconscious or in some emergency situations, informed consent is implied.

Obtaining informed consent means that patients are given an opportunity to take an

active role in the decision-making process that will affect their oral health. It provides an opportunity for patients to become informed oral healthcare consumers.

Step 1

The problem list or diagnostic summary is to be presented to patients and/or guardians in understandable terms. This will set the stage for a discussion of the patient's health status (both systemic and oral) and provide an opportunity to educate patients about the etiology, severity, and prognosis associated with each problem.

Step 2

Discuss with patients various treatment options (including the availability of additional diagnostic tests and procedures), potential benefits of the treatment recommended, possible negative outcomes of the proposed treatment, the probability of success (good outcome), and the consequences of not treating a problem.

Step 3

Inform patients of the time required to complete treatment, the cost for the services recommended, as well as the costs of alternative options. Patients have the right not only to ask questions about the costs of recommended services, but to make choices about their oral healthcare. Articulate clearly that exercising these rights also means that patients assume some responsibility for the success of the clinical process.

Step 4

To maximize the effectiveness of proposed preventive and therapeutic interventions, ensure that patients have an unequivocal understanding of their responsibility to follow the recommendations they have agreed to. It is also the patients' responsibility to provide feedback to the clinician about any problems or concerns that may arise while under treatment. In this context, the principle of "due care," patients performing their role in the clinical process, applies. Patients' failure to participate in the process, to the best of their physical and cognitive ability, constitutes negligence on their part.

Step 5

Educate patients about the dynamic nature of treatment plans. They should understand that as the sequential phases of the treatment plan are implemented, initial therapeutic interventions may provide additional data relevant to the true nature and extent of the problem, occult disease may become overt, and patient response to treatment and the effectiveness of preventive care may all mandate modification of the initial treatment plan.

Step 6

At the end of the case presentation patients should be provided an informed consent form for their signature. This is to certify that they understand the reason for the treatment; that they had an opportunity to discuss the treatment plan, including costs and alternative treatment options, with the clinician; and that they understand that there may be variations in treatment and costs if new findings are made.

Consultations and Referrals

Once a patient-doctor relationship has been established and the clinician has agreed to treat a patient, the practitioner is obligated to conduct the management of that patient's illness with "due care." Failure to render due care constitutes negligence. Negligence is the legal term for omission of care either by failure to diagnose or to adequately treat. This clearly implies that clinicians also have

Table 8.2. Reasons for consultations or referrals.

1. The diagnosis is uncertain.
2. There is doubt as to the physical and/or emotional ability of the patient to undergo and respond to dental care.
3. Managing the condition of the patient is not within the field of training of the primary clinician.
4. The primary clinician is knowledgeable about the patient's condition and its treatment but believes that a specialist is better prepared to manage the problem.
5. The patient's condition is not responding to treatment.
6. The patient or his or her agent requests a second opinion.

an obligation to seek consultation with or initiate referral to other healthcare providers whenever the welfare of the patient might be safeguarded or advanced by having recourse to those who have special skills, knowledge, and experience. Table 8.2 summarizes the various reasons why the primary clinician may initiate the consultation or referral process.

Consultation is an act of deliberation between healthcare providers related to a diagnosis or its treatment. A consultant may either be asked to give professional advice (opinion) or to provide a service. Consequently, consultation provides access to expert knowledge, sophisticated procedures, quality patient management, and patient reassurance.

Role of a Consultant in the Consultation Process

If the request is for professional advice (opinion), the authority for the patient's management is retained by the primary clinician. He or she retains full responsibility, including legal, for the welfare of the patient. The consultant assumes no direct authority in the management of the patient and is not required to write orders.

Role of the Consultant in the Referral Process

If the consultant is asked to provide a service, authority for the patient's management is transferred to the consultant with a mutually clear understanding of the purpose and duration of the referral. The consultant assumes full responsibility, including legal, for the patient's welfare. If the consultant believes that additional consultations are warranted, it must be communicated to the primary clinician before further action is taken.

Role of the Patient in the Consultation/Referral Process

The patient is an interested party in the consultation or referral process, but the choice of the consultant should not be left entirely up to the patient. Inform the patient of the reason for the consultation. Brief the patient regarding events that may occur while the patient is in the care of the consultant. Provide the patient insight into the mannerisms and personality of the consultant. Establish uniform definitions that are understood by all (the primary clinician, the consultant, and the patient).

Role of the Primary Clinician in the Consultation and Referral Process

Initiating the Consultation or Referral Process

The office of the primary clinician must coordinate the appointment with the consultant and ensure that the patient has an appointment and that it is scheduled in an appropriate time frame. In routine situations there is no particular immediacy. The appointment may be scheduled at the convenience of both the consultant and the patient.

In urgent situations, a diagnosis should be established and/or treatment initiated fairly rapidly. The patient should be seen in a

matter of days. In emergency situations, the gravity of the problem must be clearly explained to the patient and the consultant. The patient should be seen within a matter of hours.

Monitoring the Consultation and Referral Process

The primary clinician has an obligation to monitor the status of the consultation and referral process. A patient may not show up for the scheduled appointment. Likewise, the consultant may be slow in rendering an opinion or in providing a summary of services rendered. When there is a difference of opinion between the primary clinician and the consultant, the problem should be resolved out of earshot of the patient. If there is strong disagreement concerning the diagnosis or the proposed treatment, the patient must be given both opinions and an option for further consultation.

Documenting the Consultation and Referral Process

In the consultation and referral process, the request from the primary clinician and the opinion rendered or a summary of services provided by the consultant should be in writing. The consultation document is an official permanent record. While written as a confidential doctor-to-doctor communication, the consultation document is also available to the patient and others (e.g., peers and insurance companies) for review. Proper utilization and preservation of information in the consultation process are ensured by appropriate documentation methods, which, as in all aspects of the clinical process, should follow the problem-oriented method of record keeping.

Conclusion

Privileges given to clinicians by society and by patients are quite remarkable. Clinicians are permitted to ask searching personal questions, listen to personal secrets, and touch, manipulate, and explore another individual's body. It is evident that a clinician with proper credentials from society, and consent from a patient, is permitted actions accorded no other individual. With these privileges comes the responsibility to think clearly (professionalism, clinical judgment), act decisively (timely diagnosis and treatment), and care tenderly (sensitive to and considerate of patients' feelings).

The combination of privilege and responsibility mandates the establishment of a patient-doctor relationship that is to clearly benefit the patient and not one that is disguised as a means of rewarding a clinician's own need for approval or advantage. The characteristic that distinguishes, promotes, and maintains a healthy patient-doctor relationship is adherence to the principles of (1) "duty of disclosure" by the clinician; (2) "self-determination" by the patient; and (3) "due care" in the clinical process, both by the clinician and the patient.

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